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ON POISONS.

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ON

July 1860

POISONS

IN RELATION TO

MEDICAL JURISPRUDENCE

AND

MEDICINE.

BY

ALFRED SWAINE TAYLOR, M.D., F.R.S.,

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS; HON. M.D. UNIV. ST. ANDREWS; MEMBER OF THE ROYAL COLLEGE
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ROYAL COLLEGE OF VETERINARY SURGEONS; AND AUTHOR
OF A "TREATISE ON MEDICAL JURISPRUDENCE."

Das Wissen wird durch suchen sich entfalten.

SECOND AMERICAN
FROM THE
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PREFACE

TO

THE SECOND EDITION.

IN this new edition of my work on POISONS I have been compelled to restrict my remarks to a consideration of those substances which give rise to medico-legal inquiries. TOXICOLOGY is a wide subject in itself, and it would be hopeless on the part of a writer to endeavor to include in one small volume all those facts and principles which are now comprehended under this department of medical science. One of two courses is open to him,—he may give a distinct notice of every substance which has been known to have a noxious action on the animal body, and in this case the book would become a catalogue of drugs, with a brief history of poisons,—or he may exclude those substances which belong rather to the history than the practice of the subject, and thus devote more space to the consideration of substances which, from the frequency of their employment for murder and suicide, are of great practical importance. I have chosen the latter course. My space has been limited, and I have endeavored to fill it with materials which may be of profit to the practitioners of law and medicine, for whose especial use this volume is intended. Under this view the plan of the former edition has been entirely changed. Many chapters have been struck out, and an equal number of new chapters introduced. The requirements of a period dating no longer ago than ten years are different from those of the present day; and it is the duty of an author, so far as it may be in his power and consistent with the scope of his labors, to fulfil these requirements by an entire remodelling of his subject.

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No one can draw a definite boundary between a poison and a medicine. The greater number of poisons are useful medicines when properly employed, and nearly every substance in the catalogue of medicines may be converted into an instrument of death if improperly administered. For this reason it must not be supposed that a substance is not a poison because it does not find a place in this second edition of my work. I have simply exercised a freedom of selection, with a special view to practice, omitting a notice of those medicines or poisons, as the case may be, that have not hitherto been made a subject of investigation before our legal tribunals.

15 ST. JAMES'S TERRACE, REGENT'S PARK,
January 10th, 1859.

TABLE OF CONTENTS.

PAGE

CHAPTERS I to VII.—The Nature of Poisons—Their Mode of Action—Absorption—Influence of Habit—Classification, &c., ... 17–102

CHAPTERS VIII and IX.—Evidence of Poisoning in the living Body—Diseases resembling Poisoning, ... 102–148

CHAPTERS X to XIV.—Evidence of Poisoning in the dead Body—Evidence from Chemical Analysis—Experiments on Animals—Statistics of Poisoning, ... 149–222

IRRITANT POISONS.

CHAPTERS XV to XXI.—Mineral Acids—Oxalic Acid—Alkalies and Alkaline Salts—Phosphorus, ... 223–319

CHAPTERS XXII to XXXII.—Arsenic—Mercury—Lead—Copper—Antimony—Zinc and other Metals—Vegetable and Animal Irritants, ... 319–517

NEUROTIC POISONS.

CHAPTERS XXXIII to XLI.—Narcotic or Cerebral Poisons—Opium—Prussic Acid—Alcohol—Chloroform—Camphor—Tobacco—Cocculus Indicus—Darnel—Mushrooms—Henbane, ... 518–671

CHAPTER XLII.—Spinal Poisons—Nux Vomica—Strychnia—Brucia, . 672–705

CHAPTER XLIII.—Cerebro-Spinal Poisons—Hemlock—Œnanthe—Monkshood—Belladonna—Lobelia—Foxglove—Thornapple—Laburnum—Yew, . 706–742

APPENDIX, ... 743

INDEX, ... 745

CORRIGENDA.

Page 73, last line, for “weights” read “weight.”
“ 140, line 4 from top, for “*two*” read “*three*.” [The case referred to occurred in December, 1857, and the particulars were communicated to me in March, 1858.]
“ 202, line 10 from top, for “Woorara” read “Curara.”
“ 202, line 16 from top, for “of” read “for.”
“ 211, line 19 from foot, for “five grains” read “four grains.”
“ 213, line 3 from top, for “to be” read “to have been.”
“ 268, line 12 from top, for “the form” read “their form.”
“ 292, line 9 from foot, for “Schossberger” read “Schlossberger.’
“ 349, line 8 from top, for “grains” read “grain.”

ON POISONS.

CHAPTER I.

TOXICOLOGY — MEANING OF THE TERM POISON — SALINE MEDICINES POISONOUS IN LARGE DOSES — COMMON SALT AND EPSOM SALT — WHITE HELLEBORE — HIERA PICRA — OIL OF TURPENTINE — DEADLY POISON — SAL VOLATILE — GREEN VITRIOL — MEDICAL DEFINITION — LEGAL DEFINITION.

By Toxicology (derived from τοξικὸν, *poison*, and λόγος, *discourse*) we are to understand that branch of medical science which relates to the history and properties of poisons, and of their effects upon the living body. This subject is commonly regarded and treated as a part of Medical Jurisprudence; but the number and importance of the facts connected with poisons which have been accumulated of late years, have justly contributed to raise Toxicology to the rank of a distinct science. To the physician, the pathologist, and the medical jurist, a knowledge of the subject is of great importance: for cases are continually presenting themselves in which a practical application of the principles of this science is demanded; as, for example, in the treatment of a person laboring under the effects of poison, in drawing a clear distinction between changes produced in the body by disease and those caused by poison, or finally, in aiding the criminal law in convicting those who have been guilty of the crime of poisoning.

Definition.—A Poison is commonly defined to be a substance which, when administered in *small quantity*, is capable of acting deleteriously on the body; and, in popular language, it is confined to substances which destroy life in small doses. It is obvious that the above definition is too restricted for the purpose of medical jurisprudence. It would, if admitted, exclude a large class of substances, the poisonous properties of which cannot be disputed; as, for example, the salts of copper, tin, zinc, lead, and antimony, which, generally speaking, act only as poisons when administered in large doses. It must not be supposed, however, from this statement, that the compounds of these metals cease to be poisonous when exhibited in small doses. We may admit the general truth of the doctrine, that a poison in a small dose is a medicine, and a medicine in a large dose is a poison; but a medicine, such as tartarized antimony, may be easily converted into a poison, either by giving it in small doses at short intervals under states of the body not adapted to receive it, or in cases in which it exerts an injuriously depressing effect. More than one death has been lately occasioned by this wilful misuse of antimony in doses which might be described as *medicinal*, although in the cases referred to, no other intention could have existed in the secret administration of this substance than that of destroying life. A person may die either from a large dose given at once, or from a number of small doses given at such intervals that the system cannot recover from the effects of one before another is administered. In cases of lead-poisoning it is a well-known fact, that a quantity of carbonate of lead so small as to be scarcely appreciable to tests, may, by its daily introduction into the system through water or other articles of food, produce symptoms of chronic poisoning, which, although different in their nature and progress, are not less fatal than those which are produced by a large dose of a salt of lead. Water containing only one grain of lead in a gallon, or one seventy-thousandth part by weight, has thus been known to produce all the effects of lead-poisoning in an aggravated degree. Some substances, such as nitre, have not been known to act as poisons, except when taken in very large doses, while arsenic acts as a poison in small doses. But, in a medico-legal view, whether a person die from the effects of half an ounce of nitre or of two grains of arsenic, the responsibility of a person, who criminally administers the substance, is the same. Each substance must be regarded as a poison, differing from the other only in its degree of activity, and perhaps in its mode of operation. The result is the same: death is caused by the substance taken, and the *quantity* required to destroy life cannot therefore be made a ground for distinguishing a poisonous from a non-poisonous substance. If, then, a medical witness be asked, "What is a poison?" he must beware of adopting this common definition, or of confining the term poison to a substance which is capable of operating as such in a *small* dose given at once.

Saline substances poisonous.—The fact that a poison has been commonly regarded as a substance which produces serious effects when taken in small quantity, has induced many who have adopted this arbitrary view to assert, that certain substances which have actually been known to cause death, are not poisons; and this doctrine has been apparently supported by the fact, that were not some such distinction adopted, it would be difficult to separate the class of poisons from bodies which are reputed inert. In answer to this view, it is perhaps sufficient to show, that there is no good reason for assuming this as the distinguishing character of a poison; for it is impossible, even among substances universally admitted to be poisonous, to make any division according to the effects produced by the quantity taken. In relation to the quantity required to operate fatally, the difference is not so great between nitre and oxalic acid as between oxalic acid and strychnia. If we consider nitre to be a poison, there seems to be no good reason for excluding common salt (the chloride of sodium). Medical practitioners would scarcely be prepared to admit this last-mentioned substance into the class of poisons; but it is to be observed, that in a large dose it is capable of acting as a powerful irritant, and of inflaming the mucous membrane of the alimentary canal to the same extent as much smaller doses of other well-known irritants. An instance of COMMON SALT having caused death occurred in the north of England in the year 1839. A young lady swallowed, it is supposed, about half a pound of this substance, for the purpose of destroying worms. It was considered to be a harmless substance, according to the common notion; but in the course of about two hours some alarming symptoms made their appearance, and medical assistance was sent for. She was found to be in a state of general paralysis; and although the stomach-pump and other antidotal means were speedily employed, she died in the course of a few hours. After death there were found those changes in the body which are generally indicative of the effects of a violent irritant on the alimentary passages. (Medical Gazette, 1839–40, i, 559.) This case is deserving of attention, from the evidence which it furnishes of the fallacy of the popular doctrine, that what is taken so freely in small quantities, with benefit, may be taken, with equal impunity, in large doses. Dr. Christison mentions a case in which a man swallowed a pound of salt, and died within twenty-four hours, under all the symptoms of irritant poisoning. The stomach and intestines were found in a high state of inflammation after death. In another case serious symptoms were produced in a young man by a much smaller dose. In this instance the individual had taken about *two ounces* as an emetic dissolved in a small quantity of water. He was seized with an acute burning pain in the stomach, tenderness in this organ, and great anxiety, without any vomiting, until he had drank a large quantity of warm water as a remedy. Before Dr. Christison saw him he had vomited freely, but he still suffered severe intermitting pain. (On Poisons, 658.) In one instance, in which about a table-spoonful of salt had been taken by mistake for sugar, there was no vomiting or purging, but great pain in the region of the stomach, with dryness of the fauces, which lasted several days. In a toxicological view, it is not easy to distinguish the effects of common salt in these cases from the poisonous action of salt of sorrel, which, as it is well known, may be taken with impunity in small quantity, and although the designation "poison" may appear inappropriate, it would be obviously inconsistent to apply this term to one substance and refuse it to the other, when both are innocent in small, and noxious only in large doses. (See post, BINOXALATE OF POTASH.)

There is another substance commonly reputed to be innocent, but which in a large dose may destroy life. This is the sulphate of magnesia, or EPSOM SALT. A trial took place at the Huntingdon Autumn Assizes, 1842, in which two men were indicted for feloniously killing one Daniel Cox, by administering to him a large quantity of Epsom salts dissolved in beer. The deceased was an old man and a confirmed drunkard, and he was in the habit of drinking beer to excess. On the day laid in the indictment, the deceased had drunk several pints of beer, which, it was afterwards proved, had been drugged with the sulphate of magnesia. He was seized with violent purging, and died within forty-eight hours.

On an examination of the body, the lining membrane of the alimentary canal was found inflamed, and there was no doubt that death was owing to the irritant effects of the salt. One of the prisoners was convicted. The quantity of the substance taken in this case could not be ascertained, but there was reason to suppose that the dose was large.

A case is reported in which a boy, ten years old, had two ounces of Epsom salts administered to him medicinally by his father, as a remedy for worms. The salt was taken partly dissolved in a teacupful of water, and very soon after it had been swallowed, the boy staggered and became unwell. When seen by a medical man, half an hour afterwards, his pulse was imperceptible, his breathing slow and difficult, the whole frame in a state of great debility, and, in ten minutes more, the child died, without any other symptom of note, and particularly without any vomiting. (Christison on Poisons, 657.) It is remarkable that, in this case, there does not appear to have been any purging; and after death no morbid appearance was found in the body. SULPHATE OF POTASH and TARTARIC ACID have also caused death in large doses.

It has been suggested that substances of this kind connect the true poisons with those which are inert in regard to the economy; but they are assuredly to be regarded by the medical jurist as irritant poisons; and, as to the dose administered, it is of little moment in medicine or in law, whether one grain of one substance or one ounce of another substance be taken, provided the fatal effects be clearly traceable to the action of the particular substance on the body. This is the point to which a medical jurist must direct his attention. In Medical Jurisprudence, therefore, it is necessary to look to the noxious effects produced by particular substances on the system, and the adequacy of these substances to cause death under symptoms of poisoning, rather than to the mere quantities in which they may have been taken.

These remarks on the looseness of the popular definition of the term poison, have been suggested by the fact that medical men have been sometimes severely pressed in cross-examination on trials for certain criminal offences, to state what is strictly a poison and what is not. In charges of attempted poisoning, or of attempted abortion by the administration of drugs, it is by no means an indifferent matter for a witness to be able to say what substances are noxious and what are inert; or to show, how some bodies commonly reputed inert, may under certain conditions act deleteriously on the system. The conviction of a prisoner may actually depend upon the answer returned by a medical witness to a question of this kind. In this point of view a case which was tried at the Norwich Lent Assizes, April, 1846, is well deserving of the attention of medical practitioners. A woman, named Whisker, was charged with administering to the prosecutrix, a small portion of White Hellebore (Veratrum album), for the purpose of producing abortion. When the fact of administration had been clearly proved, an objection was taken to the indictment, on the ground that there was no medical evidence to show that Hellebore was a virulent poision. One medical witness is reported to have said, that Hellebore was noxious to the system and produced ill effects, but he knew of no case in which it had produced death. Under these circumstances he thought he was not justified in calling it a poison. The learned judge who tried the case said that that was a poisonous drug which in common parlance was generally understood and taken to be such; and the evidence for the prosecution he thought was sufficiently strong to bring Hellebore within the meaning of the statute. The jury returned a verdict of guilty, and in answer to the judge said that they considered Hellebore to be a poison. The remarkable circumstance in this case is, that any medical doubt should have been entertained on the subject. Every toxicologist of repute describes White Hellebore as a poison. It is not so active as many of the vegetable narcotico-irritants, but nevertheless it owes its properties to the presence of a poisonous alkaloid (veratria) which is diffused through the plant, and renders it poisonous.

Blue Vitriol or sulphate of copper is not unfrequently employed for the purposes of abortion. Is this a poison? Hitherto toxicologists have regarded it in this light, but Dr. Hoenerkopf has recently questioned the accuracy of this view, and he quotes cases in which he has given it in large doses without causing death or producing even alarming symptoms. (Casper's Vierteljahrschrift, 1855, B. viii, p. 222.) Dr. Spielmann objects to this inference on the ground that as the substance was used medicinally, and a tolerance of medicines is often set up in diseased conditions of the system, the facts recorded by Hoenerkopf, are not adverse to the views adopted by Orfila and others, namely, that blue vitriol is a poison, and, if it does not cause death, it is capable of producing injury to health. Its emetics powers are well known, and a mineral substance which produces violent vomiting, must be regarded as noxious to a pregnant female. (Casper's Vierteljahrschrift, 1856, B. x, p. 41.)

The reader will perceive from a case above quoted, that whether a particular substance is, or is not a poison, is a question of fact left for the decision of a jury from the medical evidence given in the case. In general the indictment contains a clause describing the substance as a poison or "*destructive thing*" (post, p. 26), a form of expression which should always be adopted, in order to prevent the occurrence of these technical objections. The question what is a noxious or destructive thing frequently arises on charges of attempted abortion. In the case of White (Aylesbury Lent Assizes, 1857), it was proved that the prisoner had administered to the prosecutrix a large dose of a substance popularly known under the name of *hiera picra.* This is a compound of aloes and canella bark. It was admitted to be a proper medicine in small doses, but capable of acting with injurious effects upon pregnant females when given in large doses. The prisoner was convicted. In the case of Reg. v. Rodanbosh (Central Criminal Court, December, 1856) the prisoner was indicted for administering a quantity of *oil of turpentine* to her infant with intent to murder it. That she had poured this liquid down the child's throat was clearly proved. The child suffered some time from the effects, but ultimately recovered. The defence was that the child had a cough, and turpentine was given by the mother to cure this! The prisoner was acquitted. Oil of turpentine must be regarded according to the dose as noxious to an infant. (See p. 28, post.)

Deadly poison.—There is another point of view in which this question may require to be considered, namely, What is to be understood by a *deadly* poison? In indictments for poisoning, it is customary to describe every poison as *deadly*. The substance administered might with equal propriety be described as poisonous, or of a destructive nature; but those who draw up indictments are but little informed on such matters, and they can never speak of a poison without describing it as deadly. The following case occurred on the Norfolk Spring Circuit, 1836. Two persons were capitally indicted for having feloniously caused to be administered to the prosecutor, a quantity of a certain "*deadly poison*," called sulphate of copper (blue vitriol), with intent to murder him. It appeared in evidence that all the parties were servants in a farmer's family, and that it was the duty of one of the prisoners to prepare breakfast for the other servants. On the morning of the day laid in the indictment, the prosecutor observed that the milk which had been prepared for him was very nauseous, and, after having taken a small quantity of it, he laid it aside. He was soon seized with violent vomiting, but under medical assistance he recovered. The residue of the milk was analyzed, and was found to contain sulphate of copper. In the defence the counsel for the prisoners contended that they could not be convicted of the crime charged in the indictment, since, according to all medical experience, the sulphate of copper was not a *deadly* poison. The medical witnesses, of whom there were two, were then required to give their opinions, but they differed on the point. One, a surgeon of some years' standing, considered it to be a deadly poison, although he admitted that so far as his own experience went, he had had no knowledge of its poisonous effects. The other stated that it was not a deadly poison, and that when sold in a shop, the word poison was never attached to the label. The judge considered the case to be one of suspicion rather than of proof, and the prisoners were acquitted. Although by this summary disposal of the case, the force of the objection to the indictment was rather evaded than decided, yet the difference of opinion between the two medical witnesses is worthy of remark. It appears to me that the term *deadly* should be applied to those poisons only which prove speedily fatal in small doses, such as strychnia, morphia, aconitina, nicotina, prussic acid, and arsenic; it cannot with propriety be applied to such substances as the sulphates of copper and iron, or, as in the subjoined case, to such a liquid as "spirits of hartshorn." In this case (Reg. v. Haydon), which was tried at the Somerset Spring Assizes, 1845, common sal volatile was absurdly described as a "deadly poison." The counsel for the prisoner took an objection to the indictment on this ground, but the judge (Erle, J.) said, "the word *deadly* appears to me to be used merely in pursuance of an ancient form, and not to be essential to the validity of the indictment. It would be sufficient to describe it simply as a poison, and under that term would fall, *anything calculated to destroy life*. Substances harmless in themselves might become poisons by the time or manner of their administration. This seems to me the view most accordant with common sense, and therefore I hold this indictment to be good, even although it describes spirits of hartshorn as *deadly poison*." (Law Times, April 12, 1845.) The question may, by this decision, therefore, be considered as settled. The word *deadly* must henceforth be regarded and treated as mere surplusage.

A similar question arose in a trial which took place at Chelmsford some years ago, where the substance administered was copperas or sulphate of iron. A man was charged with having administered this substance to two women, with intent to murder them; and in the indictment it was described as a *deadly* poison. The medical witness stated that it was not, properly speaking, a deadly poison. This is so far true, that it is rare to meet with a case in which this substance has destroyed life. There is no doubt that green vitriol is capable of acting as an irritant, and as such, if taken in a large dose, and not ejected from the stomach by vomiting, it may produce inflammation of the viscera and death, but it certainly has no claim to be considered or described as a "deadly" poison, and, in fact, some authorities exclude it from the class of poisons altogether. A trial took place at the Assizes for the Seine, September 11, 1848, in which a medical opinion on this subject was necessary in order to support the charge against the accused. A man, without any apparent motive, administered this substance to his wife at various times. She suffered from severe vomiting and purging, and she appeared to her medical attendant to be in a dangerous condition, but she shortly recovered. M. Chevallier proved that the substance administered was copperas or green vitriol. At the trial one medical witness said it was noxious or injurious to health, but not a poisonous salt; while another did not consider it dangerous, because it would be impossible for a person to take a sufficient quantity to cause death. M. Chevallier stated that there was no well-established case of poisoning by it on record;—that experiments on animals in which the gullets had been tied were not satisfactory, and that a woman to whom large doses of it had been administered for several days with criminal design, had not died from the effects. The President left it to the jury, whether the symptoms suffered by the wife had been caused by the wilful administration of a substance which, although it might not cause death, was injurious to health (*nuisible à la santé*). They answered in the affirmative, finding the defendant guilty, and he was sentenced to five years' imprisonment. (Annales d'Hygiène, 1850–51, 180.) By reference to the section on SULPHATE OF IRON (post), it will be seen that there have been instances in this country in which it has proved fatal to life. Some years since I was consulted on the poisonous nature of this salt as employed in the adulteration of beer. The answer given was that, admitting it not to be a poison according to the popular meaning of the term, the daily administration of it would prove noxious to health.

As the greater number of substances known under the name of medicines may act like poisons, according to the dose or circumstances under which they are administered, and no precise boundary can be laid down, it would seem that the proof of the crime of poisoning should rest upon the *intention* with which the substance is administered and on the effects produced, or on satisfactory evidence, that it is capable either of destroying life or of causing injury to health. A man may administer a substance, such as tartar emetic, in *medicinal doses* with good or evil intention. His intention, which is a question for a jury, may be not to remove disease, but to destroy life. He may administer it secretly under circumstances in which its lawful use would certainly not be required;

he may continue to use it at intervals, in medicinal doses, even when its dangerous effects are clearly manifested by symptoms, and when any medical man, dealing *bonâ fide* with a patient, would immediately withdraw it as a medicine. Is such an act as this to be covered by that thin cloak of medical sophistry which was spread over it in the case of William Palmer? Is it to be received as a reasonable or safe doctrine for society, that tartar emetic is a *medicine* and *not* a *poison;* that it has been given in some cases in large doses without causing death; and that, provided the doses are what may be called "medicinal," there can be no poisoning? If a criminal act is thus frittered away by a definition, murder by poisoning might be carried on throughout this country with impunity whenever a motive for crime presented itself; and the greater the skill and the more perfect the cunning used in its perpetration, the more surely might a criminal rely on receiving a certain amount of quasi-professional protection. M. Bernard, in remarking upon the supposed distinctions between medicines and poisons, observes, that a substance which is a medicine in a small dose may act as a poison in a large dose, or if given at an improper time. A correct definition of a poison is impossible, and it is so far satisfactory that those subjects which are the most difficult to be defined, stand the least in need of a definition. (Leçons sur les Effets des Substances Toxiques, &c., par Claude Bernard, p. 39, 1857.)

Medical definition.—In legal medicine, it is difficult to give such a definition of a poison as shall be entirely free from objection. Perhaps the most comprehensive definition which can be suggested is this: "A poison is a substance which, when introduced into the stomach or bowels, is capable of destroying life without acting mechanically on the system." But it is well known that some substances act as poisons by absorption when applied to the skin, or a wounded or ulcerated surface; while others, again, as the poison of the viper, and of rabies, may have their fatal effects limited to those cases in which they are introduced by a wound: and a third class may destroy life merely by their chemical effects upon the parts with which they come in contact, without necessarily poisoning the blood by absorption, *e. g.* sulphuric acid. Leclerc, who wrote in 1803, defines a poison to be a substance which, when taken in a small dose internally, or applied to the living body in any way externally, is capable of injuring health or causing death. (Essai Médico-légale sur l'Empoisonnement, p. 49, Paris, An xi.) This definition has been substantially adopted by some modern toxicologists. It is, however, not only open to the objection that most poisons must be taken in *large* doses in order to injure health, or cause death, but it would include melted lead or boiling liquids among poisons; for these, when applied to the living body externally, injure health and cause death. M. Flandin considers those substances only to be poisonous which are capable of entering into the body and producing their effects by *absorption.* (Traité des Poisons, i, p. 193.) This definition is not sufficiently comprehensive, because it excludes from the class of poisons the mineral acids and alkalies. A similar objection appears to me to exist to the definition of a poison by the late Dr. Griffiths, the American editor of my Manual of Medical Jurisprudence: "A poison is a substance which, when taken internally, or applied to the surface of the body, is capable of destroying life without acting in a purely mechanical manner." Can it be said that boiling water, or oil, or melted lead, when applied to the surface of the body, destroys life by exercising mechanical action? If not, then these liquids are poisonous. If, however, this kind of action be regarded as mechanical, then the mineral acids and alkalies must also be considered by their effects to act mechanically, whether applied to the skin or the mucous membrane of the stomach. These remarks show that it is difficult, if not impossible, to comprise in a few words an accurate description of what, in medical language, should be understood by the term "poison."

Under the definition which I have above given, it might be objected that the whole class of medicines, and numerous substances of an inert nature, would be included. Thus it is well known, that there are many cases on record in which *cold water*, swallowed in large quantity, and in an excited state of the system, has led to the destruction of life either rapidly by shock, or slowly by inducing gastritis. Any cold liquid, such as iced water, beer, or ice itself, may have an equally fatal effect. The action of water or cold liquids, under these circumstances, cannot be said to be mechanical; it appears to be due to the shock suddenly induced on the nervous system through the lining membrane of the stomach, and yet it would be inconsistent to class these inert liquids among poisons. It is, however, important to notice that the effects produced by cold liquids sometimes closely resemble those caused by poison. A girl, æt. 9, during sultry weather, and while in a heated state, drank a coffee-cupful of cold water. She immediately fell to the ground in a state of insensibility. When seen by a medical man, half an hour afterwards, she was quite unconscious: the skin was cold, the pulse feeble, and the pupils were unaffected by light. There were also convulsive twitchings about the corners of the mouth. She was bled, stimulants were applied, and in five or six hours she recovered. (Lancet, Oct. 7, 1843.) The reader will find this subject treated in its medico-legal bearings by Dr. Guerard, in the Annales d'Hygiène, for 1842, tome i, p. 42.

In all cases of this description, it appears to me that we are justified in drawing the following distinction between poisonous and non-poisonous substances. If the deleterious effect does not depend upon the nature of the substance taken, but upon the state of the system at the time at which it is swallowed, the substance cannot be regarded as a poison. All poisonous substances are *per se* deleterious,—the state of the system, setting aside for the present the peculiar effects of idiosyncrasy and habit, has very little influence on their operation. The symptoms may be suspended for a time or slightly modified in their progress, but sooner or later the poison will affect the healthy and diseased, the old and the young, with a uniformity in its effects not to be easily mistaken. A distinction of this kind cannot, however, be drawn, except by a professional man who has given attention to the subject of toxicology; and therefore it is no matter of surprise that poisoning should have been in more than one instance erroneously imputed in cases where death has followed the drinking of cold liquids.

Legal definition.—In reference to the medical definition of a poison it is necessary to observe, that the law does not regard the manner in which the substance administered acts. If it be capable of destroying life, or of injuring the health of an individual, it is of little consequence, so far as the responsibility of a prisoner is concerned, whether its action on the body be of a mechanical or chemical nature. Thus a substance which simply acts mechanically on the stomach, may, if wilfully administered with intent to injure, involve a person in a criminal charge, as much as if he had administered arsenic or any of the ordinary poisons. It is then necessary that we should consider what the law strictly means by the act of poisoning. If the substance criminally administered destroy life, whatever may be its nature or mode of operation, the accused is tried on a charge of murder or manslaughter, and the duty of a medical witness consists in showing that the substance taken was the certain cause of death. If, however, death be not a consequence, then the accused is tried under a particular statute for the attempt to murder by poison. (1 Vict. c. 85, sec. 2.) The words of this statute are very general, and embrace all kinds of substances, whether they be popularly or professionally regarded as poisons or not. Thus it is laid down, that—

"Whoever shall administer, or cause to be taken by any person, any poison, or other destructive thing, with intent to commit murder, shall be guilty of felony, and being convicted thereof, shall suffer death."

Although the administering be followed not by death but only by bodily injury dangerous to life, it is still a capital felony, providing the *intent* has been to commit murder. The *attempt* to administer to any person any *poison*, or other destructive thing, with like intent, &c., although no bodily injury be effected, is felony, punishable by transportation for life, for fifteen years, or imprisonment for any term not exceeding three years. From the words of this statute it appears that the law requires, in order to constitute the crime of poisoning, that the substance should be *administered to*, or be *taken by*, an individual. Several deaths have been caused of late years by the external application of arsenic and corrosive sublimate to ulcerated and diseased surfaces. Supposing the poison is thus applied intentionally, and great bodily injury is done to an individual, it might be a question whether the crime could be punished under these sections of the statute. Lord Campbell's act (14 and 15 Vict. c. 19) appears to provide for this description of offence, although the application or administration is herein limited to chloroform, laudanum, or other stupefying drug. The external application of arsenic so that it produces personal injury, would no doubt be considered an act of administration.

It will be perceived that the words of the statute leave the question, "what is a poison," to depend upon the medical evidence adduced. In a trial which took place at the Chelmsford Assizes, a woman was charged with administering *white precipitate* to her husband with intent to kill. She was acquitted on the ground that there was no evidence to show that white precipitate was either a poison or a destructive thing. It is, however, placed beyond doubt that this substance is not only capable of producing noxious effects, but of destroying human life; hence, this acquittal was based on a pure mistake. White precipitate is not by any means so poisonous as corrosive sublimate, but it is undoubtedly a mercurial poison. Unless the medical evidence received by a court when this question is raised, be very closely investigated, a great mistake may be made, owing perhaps to want of experience or want of reflection on the part of the witness to whom the question is put.

This question, "What is a poison?" may present itself under another aspect. In the Queen v. Cluderay (Exchequer Chamber, January 19, 1849), the prisoner was indicted for administering poison with intent to murder. He was proved to have administered to a child, nine weeks' old, two berries in the husk of Cocculus Indicus, and the berries passed through the body of the child without doing any injury. It was submitted for the prisoner, that being in the husk they could not be considered a poison. The point was reserved by Mr. Justice Williams, who tried the case at York. It was now contended for the prisoner, that although the kernel of this nut was poisonous, still having been given in the husk, which was hard of digestion, it could not be considered as an administering of poison within the statute 1 Vict. c. 85. The Chief Justice said, the Court was of opinion, that when a man administered something that was poison with intent to murder, but in such a way that it did not act, he was guilty. Conviction affirmed. This is the only reasonable view to take of such an objection. The seed contains the poison, but the husk is inert; nevertheless the berry taken as a whole must be regarded as a poison.

The actual quantity of a poisonous substance found in an article of food does not affect the culpability of a person indicted for administering it. In the case of Hartley (Central Criminal Court, May 12, 1850), in which an attempt was made to administer sulphuric acid mixed with coffee, Mr. Justice Cresswell stated: If poison be administered with intent to murder, it is not necessary there should be enough in the article administered to cause death. If any poison be there, and the intent be proved, the crime of attempting to administer poison is complete. In the case of Reg. v. Southgate (Chelmsford Lent Assizes, 1849), Baron Parke said, in reply to an objection taken, it was quite immaterial to define or prove in what vehicle a poison was given, or whether it was administered in a solid or liquid form.

CHAPTER II.

MECHANICAL IRRITANTS—MEANING OF THE WORDS DESTRUCTIVE THING —MERCURY—METALLIC IRON—SWALLOWING COIN—PINS AND NEEDLES —MODE OF ACTION OF MECHANICAL IRRITANTS—SPONGE—HAIR— PERCUSSION CAPS—POUNDED GLASS—BOILING LIQUIDS.

Mechanical irritants.—The present state of the law in respect to administering or attempting to administer poison with intent to murder, has been already fully considered. While the words of the statute render it unnecessary for a medical witness in such cases to give judicially a close definition of "a poison," they impose upon him a difficulty which he must be prepared to meet. The substance administered may not be a poison in the medical signification of the term, nor may it be popularly considered such; and yet, when taken, it may be destructive to life. We have examples of substances of this description in iron filings, powdered glass, sponge, pins and needles, and such like bodies, all of which have been administered with the wilful design of injuring, and have on various occasions given rise to criminal charges. In cases of this kind, the legal guilt of a prisoner may often depend on the meaning assigned by a medical witness to the word *destructive thing.* Thus, to take an example: Liquid mercury might be poured down the throat of an infant, with the deliberate intention to destroy it. A question of a purely medical nature will then arise, whether mercury be "a destructive thing" or not; and the conviction of the accused will probably depend on the answer returned by the witness. Should a difference of opinion exist—an occurrence by no means unusual in medical evidence—the prisoner will, according to the humane principle of our law, receive the benefit of the doubt. The point which here requires to be considered is, why any difference of opinion should exist among witnesses.

With regard to the case just supposed, it is a general principle in toxicology, that the pure metals are not poisonous; and they are not to be regarded as "things" destructive to life, unless the mechanical form in which they are taken be such as to injure the viscera with which they may come in contact, leading to inflammation and its consequences. Even where the mechanical form favors the production of these serious results, especially perforation of the intestines, the powers of nature are often exerted in a most extraordinary way, and the individual survives apparently in good health. This has been witnessed in the case of many who have swallowed knives or pins and needles. The escape of such persons must, however, be regarded as the result of accident. They are always in imminent danger, and they commonly die sooner or later from inflammation or perforation of the viscera. (For a remarkable case in which death took place from this cause in Guy's Hospital, see Medical and Physical Journal, October 1809, p. 350.) The masses of iron which caused death in this instance are preserved in the Museum collection. (For another case in which death took place from disease of the brain, and a large number of iron nails and other metallic substances were found in the stomach, see Dublin Medical Journal, September, 1835; also Med. Gaz. xvi, 791.)

Liquid *mercury*, the substance which we have here taken as an illustration, cannot operate deleteriously on the body either chemically or mechanically. It may be taken, and has often been swallowed in considerable quantity, without perceptibly affecting the health. If a medical witness were not aware of these facts, and did not sufficiently reflect upon the nature of the question addressed to him, he might improperly cause the conviction of the accused person. The intention of the accused may have been criminal, but that is a matter unconnected with the duties of a witness: he is simply required to state whether the means employed to carry out this criminal intention, were such as were likely to produce danger to life. Similar observations might be made with regard to numerous other substances employed in medicine or in the arts; and it is quite obvious that difficulties of this kind can only be properly met by those practitioners who have closely attended to the subject of toxicology.

It is well known that bodies which are not in their own nature destructive, may become so through indirect causes. *Metallic iron* is not a poison, nor can it, except under certain circumstances be regarded as a destructive thing. An angular mass of iron may destroy life by perforating the viscera; or, if the metal be exhibited in the state of filings in doses frequently repeated, then it may become a source of irritation in the stomach, and lead to ulceration and perforation. There will be no difficulty, however, in forming an opinion on this and similar cases. Sometimes the substance may be of a nature to produce a poisonous compound in the alimentary canal. Metallic arsenic is not considered to be poisonous, but it is capable of forming arsenious acid when in a finely divided state, and thus leading to death. Thus metallic arsenic, if not considered to be a poison, must still be regarded as a "destructive thing." The metal *copper* may act on the system either mechanically or chemically: when in the stomach or bowels it may cause death by ulceration, or it may produce, with the acid and mucous liquids, the subchloride, acetate, or carbonate of copper. Sometimes the metal may acquire a coating of sulphuret, in which case its operation would be mechanical, since the sulphuret of copper is not poisonous. The rapidly destructive effects of these metallic substances, when acting mechanically, are well shown by a case reported by Mr. Dicken. (Med. Gaz. xxxv, 885.) A boy, aged ten years, accidentally swallowed a copper halfpenny. There was very slight constitutional irritation: purgatives were administered, but the coin was not passed. There was merely a sense of weight and uneasiness in the epigastrium, with a feeling of distension, which subsided in a few hours. On the twenty-seventh day after swallowing the coin, he was suddenly seized with sickness, and instantly vomited more than a quart of arterial blood. The bleeding continued the following day, when he felt something pass from his stomach into his bowels—the hemorrhage recurred, and he died on the twenty-ninth day. On opening his stomach, a circular patch of ulceration was observed on the mucous membrane, at a considerable distance from the intestinal opening; but there was no appearance of inflammation. The coin was found at the termination of the large intestines, lying loose and easily removable. There was no morbid appearance of any part of the alimentary canal, except the circular ulcer in the stomach (probably caused by the coin), which had led to death by hemorrhage. The coin had not undergone any chemical change. Gold, silver, and tin have been occasionally swallowed. These metals can only act mechanically: and they have been frequently known to pass through the bowels without materially affecting health. In one instance seven half-crowns were swallowed and discharged. (See case, Medical Gazette, xx, 275.) In March, 1854, a little girl, æt. 4, swallowed accidentally a sixpence and a fourpenny piece. She felt no uneasiness nor did she complain of any pain, sickness, or other symptoms. A quantity of bread and milk was given to the child, and without any further treatment, the coins were passed by the bowels within twenty-four hours after they were swallowed. Metallic substances may be detained in the body for a very long period and yet be ultimately discharged. In February, 1856, a case was communicated to the Medical Society of London, in which a silver dessert-spoon, seven inches long, had been swallowed by a boy. It produced some irritation in the stomach and cæcum, and was not passed until after the lapse of two years. It was quite black, much corroded, and in two pieces. (Lancet, February 16, 1856.)

Among the criminal methods resorted to for the purpose of destroying the lives of infants and children, that of causing them to swallow *pins* or *needles* in their food, is one which claims the attention of medical jurists. This mode of perpetrating murder has been brought to light by the evidence given on several criminal trials, which have taken place in England and on the continent. In cases of this kind, death is commonly to be referred to inflammation: and a practitioner can have no hesitation in designating these bodies, when exhibited to young children, as "destructive things:" they are at all times likely to lead to serious injury, if not to death: and it is no answer to this view to assert, that they have been often swallowed with impunity. We know that active poisons are sometimes taken without causing death; but this does not alter our opinion, that they are substances destructive to life, and likely to give rise to the most serious consequences. A case is reported in the Medical Gazette (vol. xxvi, p. 682) which will show how far the powers of nature are sometimes capable of resisting the effects of these mechanical irritants. Here it appears that two hundred and fifty-four pins and needles were removed from a female, aged 23, in whose body the greater number had remained for a period of thirteen years. A case is reported by Dr. Neumann, in which a pin accidentally swallowed remained in the body of a young woman for a year, and was finally discharged through the skin without injurious consequences. (Casper's Wochenschrift, 1846, 180.) That death may ensue from this cause is an undoubted fact. In the Registrar's Report for 1838–9, one child is stated to have died from the effects produced by swallowing a pin. In August, 1841, a boy, aged eleven years, was committed to Newgate on a charge of theft. Shortly after his imprisonment he swallowed a quantity of pins on a trifling wager. He soon afterwards became extremely ill, and died in the course of a few weeks, evidently from the effects of the mechanical irritant he had swallowed. A girl was tried in France, in 1838, upon a charge of having endangered the life of an infant, aged five weeks, by administering to it pins. The medico-legal investigation of this case was intrusted to M. Ollivier. From the evidence given on that occasion, it appears that in the opinion of M. Ollivier, these mechanical irritants are likely to produce more serious effects in an adult than in an infant; but this view is not based upon any particular facts. He also asserted that pins and needles, when swallowed, were comparatively harmless, and that a fatal termination was the exception to the rule. (Annales d'Hygiène, t. xxi, 178.)

In regard to the opinion expressed by M. Ollivier of the non-production of serious consequences by these mechanical irritants, it may be observed that there is a sufficient number of fatal cases on record to justify us in asserting that they are destructive things, and may endanger life. The following is, in this respect, a case of some interest, since, on an examination of the body, death was clearly referable to the mechanical irritant. A female was charged with having caused the death of her child by administering to it pins. Nine days after its birth, a pin was found in its mouth; and in about six days afterwards the child died. The mother confessed that she had caused it to swallow pins for the purpose of destroying it. The child had been born prematurely, and was of weakly habit. The abdomen was carefully examined, and the transverse arch of the colon, with the small intestines near it, was in a state of inflammation. On turning over the liver, it was found to be penetrated on its under surface near the gall-bladder by a pin, the head of which could be felt within the duodenum. The liver, pancreas, and intestines were glued together by bands of adhesive matter. On laying open the cavities of the viscera, the head of the pin was found to be near the pylorus. The pin was of a large or coarse kind, about an inch and a half in length; and after traversing the parietes of the duodenum, it had penetrated into the liver for about an inch from before backwards. There were marks of inflammation in the liver, but the other viscera were healthy. Death was undoubtedly due to the inflammation produced by this mechanical irritant. (Henke, Zeitschrift der S. A. 1838.)

It is obvious that the fatal effects thus produced by pins or needles must be in a great measure accidental. It is not from the number of these articles swallowed, nor from the age of the subject, that any just inference can be drawn as to the probability of their proving fatal to life. If it be true, as M. Ollivier has asserted, that death is the exception to the rule, it is not the less true, that the life of any person who has swallowed pins or needles, is always in danger until they are discharged. Sometimes, in these cases, life may be suddenly destroyed by hemorrhage. Mr. Bell has published a case in which a young man, aged 18, accidentally swallowed a needle in soup. In the course of ten days, he had several attacks of spitting of blood, and in one of these fits he vomited a large quantity, and expired in a few minutes. On examination, a fine sewing-needle was found lying across the œsophagus, the point of which had penetrated the right common carotid artery, and had led to the fatal bleeding. (Med. Gaz. xxxi, 694.) (For an ingenious method of detecting needles in the body when buried beneath the skin, I may refer to a pamphlet of Mr. A. Smee, London, 1845.) That a medical jurist should be prepared for giving an opinion on the effects of mechanical irritants will be evident from the following case, which came to trial on a charge of murder at the Chelmsford Assizes in 1835. The prisoner, an old woman, was indicted for the murder of her granddaughter, by causing her to swallow some sponge and a piece of wood. It was suspected that she had also administered pins to the child. The deceased was eleven weeks old; and until within a very short period of its death, it had appeared to enjoy very good health. The evidence of the only medical witness examined, was to the following effect. He stated that, on an examination of the child, he had found the mucous membrane of the stomach, as well as the liver, inflamed, and there were adhesions of the peritoneum. The stomach contained a piece of wood, and there were several pieces of sponge in the large intestines. On inspecting the viscera more closely, he discovered a pin in the substance of the liver, on its convex surface next the stomach. The pin was discolored by the fluids of the body. The substances which he found in the body were sufficient to produce inflammation; and it was, in his judgment, this inflammation which had caused the death of the child. The witness could give no opinion to the question how the pin had penetrated into the liver. On cross-examination, he admitted that it may have found its way into the cavity of the abdomen by accident. The wood and sponge might also have been accidentally introduced during the dressing and feeding of the child! It was left to the jury to say, whether the substances found in the viscera, which, by mechanical irritation, had led to inflammation and death, had been introduced wilfully or accidentally, and as there was no direct evidence on this point, they acquitted the prisoner. In this case, the mechanical irritation was probably as much due to the sponge as to the pin. The quantity of sponge found within the intestines was small. It is difficult to conceive how these different substances could have been accidentally swallowed by an infant.

Sponge may be regarded as a mechanical irritant; but little is known concerning its action on the human body. In the Medical Gazette (vol. xxxi, p. 124), two cases are related in which this substance was swallowed by a horse. In one case it did not appear that the animal suffered any inconvenience; but in the other case, it became alarmingly ill. There can be but little doubt that, when sponge in large quantity remains lodged in the viscera, it is capable of producing inflammation and death. Dr. Chowne, however, met with a case in which a small piece of sponge, accidentally swallowed by an infant, produced no injurious effects.

A case is reported in the Dublin Medical Press (Aug. 19, 1846, p. 117), in which a female, æt. 19, an imbecile from childhood, had acquired the practice of swallowing portions of her own *hair*. The symptoms shortly before her death were, great emaciation, pulse small and weak, constant vomiting after taking food, which was always liquid, as the slightest solid caused excessive pain. There was a hard tumor perceptible in the pit of the stomach. The real cause of death was not even suspected, until an inspection of the body was made, when it was found that the stomach was distended by a large mass of human hair. The effects of mechanical irritants are further shown by the case of a child who suffered from purging, heat in the stomach, coldness of the extremities, and other symptoms, in consequence of her having swallowed some copper *percussion caps*. The alvine irritation disappeared after twenty-four hours; the caps were discharged and the child recovered. (Philadelphia Med. Examiner, June, 1847, 332.) A singular case is quoted by Dr. Frank, in which a child, æt. 3 years, died from the effects of the *tooth* of a viper (*Trigonocephalus*), which it had swallowed. The mucous membrane of the stomach was of a cherry-red color, and the foreign body, covered by a layer of sloughy membrane, was found near the intestinal orifice. (Casper's Wochenschrift, Aug. 14, 1847, 581.)

It is to be observed, however, of such small substances as kernels of oranges, the stones of fruit, and other indigestible solids of the like nature, that in passing through the intestines they are liable to cause death by entering into the appendix of the cæcum and there exciting inflammation and gangrene. Such cases have been erroneously referred to as irritant poisoning. The real cause of death will be apparent on a proper examination of the body.

Certain articles of food may even act mechanically and destroy life, by simple over-distension of the stomach. A singular instance of this kind, in which a quantity of *raw rice* was the immediate cause of death, occurred in December, 1846. A young woman, æt. 22, ate a tumblerful of raw rice mixed with milk, just before taking her tea. In a few hours she became suddenly ill with severe pain in the region of the stomach, and great restlessness, evidently due to the distension of this organ from the swelling of the rice by the imbibition of fluid. Emetics were given with great relief, a large quantity of rice being expelled from the stomach. The next morning the pain increased suddenly and violently, with cold extremities, small feeble pulse, and great abdominal tenderness. She died about twenty-four hours after taking the rice. On examination, the peritoneum was found extensively inflamed; there was a deposition of lymph, with a copious effusion of turbid serum. The stomach and duodenum were empty, and free from inflammation. (Case reported by Mr. Howell, Lancet, April 10, 1847.)

A case was referred to me in April, 1847, in which there was great reason to believe that a child, aged seven years, had died from the effects of a quantity of *orange-peel* with which it had gorged its stomach. It complained of severe pain in the stomach, vomited repeatedly a yellowish colored matter, became convulsed, and died in twenty-four hours. A quantity of yellow fluid with orange pips and orange-peel was found in the stomach. The mucous membrane was generally reddened, and in several places there was well-marked injection. No poison was found, nor was there reason to suspect, except from sudden death, that any had been administered. There was no other cause for death but the irritant effects of the large quantity of orange-peel eaten by the child.

Among mechanical irritants, there yet remains to be mentioned one, which was formerly regarded as an active poison, namely, *pounded glass*. Recent observations have satisfactorily shown that this substance is not a poison. It is liable to inflict injury upon the alimentary canal, just in proportion to the size and sharpness of the fragments; and whenever it is swallowed in a state of coarse powder, it may irritate and excite inflammation of the stomach and bowels. Glass, in very fine powder, is decidedly alkaline; but it does not possess any of the properties of an alkaline poison: on the contrary, in that condition in which its alkalinity is most manifested, it appears to be inert. It is said that six or seven ounces of this substance have been given to a dog without producing any inconvenience to the animal. A trial for murder occurred in Paris in 1808, in which the accused was charged with having poisoned his wife by administering to her pounded glass. This substance was found in the stomach, and both this organ and the intestines exhibited marks of great irritation. Baudelocque and Chaussier gave their opinion that the glass was not the cause of death. Portal relates an instance of a young man who, during a debauch, broke a glass between his teeth, and then swallowed some of the fragments. These were afterwards expelled by active vomiting, and he recovered. In an attempt made by an ignorant person some years since to poison a whole family with coarsely powdered bottle-glass mixed with food,—no inconvenience resulted to those who had swallowed a portion of the glass. A case is, however, reported, in which it appears highly probable that a child, aged eleven months, was killed by the administration of this substance. Powdered glass was found in the mucus of the stomach, and the lining membrane itself was very vascular. (Christison, 654.) It is obvious that a substance of this description cannot be easily swallowed by an adult, without his being perfectly aware of it; and the instances in which it has been administered to infants are few in number. Although I believe the only instance reported of its having acted fatally is in the case of the infant just described, yet a medical jurist cannot hesitate to say that pounded glass is a mechanical irritant; and that the irritation caused by the presence of a large quantity of this substance in the stomach or bowels, might lead to fatal inflammation. (For a case, in which a medico-legal question was raised on the effects of pounded glass and mechanical irritants generally, see Annales d'Hyg. 1830, i, 364.) Experiments on the action of this substance on animals have been performed by Dr. Rufz. (Annales d'Hyg. 1844, ii, 195.). There is one kind of glass of which arsenic as arsenious acid appears to be an ingredient. This is the opal glass. Dr. Pavy has separated arsenic in rather large proportion from this variety of glass, and it is obvious that the more finely it is powdered the more dangerous are its effects likely to prove. In such a case, however, the symptoms might present the characters of arsenical poisoning.

Boiling Liquids.—Some toxicologists have placed hot liquids, such as *boiling water* or *oil*, in the class of mechanical irritants; but the effects produced by such liquids cannot with propriety be said to be mechanical. They do not act like poisons, although they leave in the body changes similar to those produced by corrosive poisons. Death from the accidental swallowing of boiling water is not uncommon among children. According to the observations of Dr. Hall and Mr. Ryland, the fatal result is most commonly to be ascribed to inflammation of the fauces and larynx, produced by the contact of the boiling liquid. This inevitably leads to suffocation, unless assistance be at hand. In one case, a child was actually asphyxiated from this cause, when my colleague, Mr. E. Cock, by the timely performance of tracheotomy and inflation of the lungs, succeeded in restoring it. Sometimes, however, inflammation of the stomach is a consequence. A case of this kind occurred a few years since at Guy's Hospital, and on examination of the body the mucous membrane at the larger end of the stomach was found to be much inflamed. The appearance was very like that produced by the common mineral irritants, although it was more confined to one part of the mucous membrane. In the Registrar's Report for 1838–39, twenty-four deaths are stated to have occurred among children from this cause alone!

CHAPTER III.

ABSORPTION OF POISONS—MODIFIED BY SURFACE AND STATE OF THE POISON—PROOFS OF THEIR ENTRANCE INTO THE BLOOD—DIFFUSION AND DEPOSITION OF POISONS IN THE LIVING—CHANNELS OF ELIMINATION—ARSENIC—ORGANS IN WHICH IT IS DEPOSITED—PERIOD FOR DEPOSITION IN THE LIVER AND OTHER ORGANS—ELIMINATION BY THE URINE AND BILE—PERIOD FOR ENTIRE ELIMINATION—ANTIMONY—ABSORPTION, DEPOSITION, AND PERIOD OF ELIMINATION IN MAN AND ANIMALS—CHANNELS OF ELIMINATION.

POISONS may enter the body by various channels. The aerial poisons, including gases and vapors, enter by the air-passages during the act of respiration. Metallic and metalloidal poisons which are capable of assuming the gaseous form may also find their way into the body by the lungs. Arsenic, antimony, and phosphorus, in their combinations with hydrogen, may act as aerial poisons. In the section on arsenical poisoning will be found described some cases in which arseniuretted hydrogen has thus proved fatal. The gaseous or aerial poisons will hereafter receive special consideration.

In reference to medical jurisprudence, poisons are either solid or liquid substances, derived from the mineral, animal, or vegetable kingdom. They may be introduced into the body, 1, by contact with the broken or unbroken skin; 2, by wounded or ulcerated surfaces; 3, by the stomach; 4, by the rectum. The mucous membrane of the nose, eye, or vagina, may also be made the medium of their introduction; but in all ordinary cases of poisoning, the liquid or solid comes in contact with the lining membrane of the stomach, and it is to the effects produced under these circumstances that a medical practitioner has chiefly to direct his attention.

Absorption.—As a general rule, whatever may be the surface or texture to which a poison is applied, it is absorbed and circulated with the blood before it begins to manifest its effects. Liquid poisons when swallowed (if we except substances which have a local and corrosive action) are more rapidly absorbed than those which are solid. Soluble poisons, such as cyanide of potassium, are absorbed more rapidly than those which are insoluble; and the larger the quantity of fluid in which the soluble poison is taken, the more speedily is it carried into the circulation. Some solid substances, which are but little soluble (arsenious acid), are, however, very soon absorbed in sufficient proportion to produce well-marked symptoms. Others, which are not very soluble in water, may become dissolved in the acid secretions of the stomach, and are then absorbed. The carbonate of lead and arsenite of copper furnish instances of this kind of action.

Absorption varies in its degree and rapidity not only according to the state of the poison, but according to the nature of the surface to which it is applied. It takes place slowly through the *unbroken skin;* nevertheless I have known symptoms of incipient narcotism to arise, during the application of an ordinary opiate liniment to the back. Belladonna, creasote, prussic acid, morphia, and numerous other agents, have a well-known action by absorption through the skin. The frequent handling of lead or pewter has been known to give rise to symptoms of chronic poisoning by lead; and the effects of mercurial poisoning may be produced by the application of mercury in a finely divided state to the skin.

When the cuticle is removed, and the surface of the true skin is laid bare, then the absorption of poisons takes place with much greater rapidity. Any ulcer or wound is a ready medium for the absorption of poison. Arsenic, as well as all mineral and vegetable poisons, may thus give rise to a train of symptoms similar to those caused by the introduction of the poison into the stomach and bowels. The use of arsenic in the manufacture of candles, as well as in the preparation of arsenical greens for paper-hangings, has produced injurious effects in some instances through the unbroken skin (Annales d'Hygiène, 1847, ii, p. 67, Assoc. Med. Journal, September 20, 1856, p. 811), and the application of quack preparations containing this poison to scirrhous ulcers, has in several instances caused death with the usual symptoms. Sometimes the substance itself acts chemically upon the skin, removes the cuticle, and thus leads to complete absorption. Solutions of bichromate of potash and cyanide of potassium have by their corrosive action given rise to symptoms of poisoning. Absorption occurs with great rapidity when the poison is introduced into the *cellular tissue* beneath the skin; and small quantities have then been observed to produce powerful effects. Thus Orfila found that from a grain to a grain and a half of arsenic in fine powder was sufficient to kill a dog under these circumstances. (Toxicologie, 1852, 5ème éd. p. 429.) This is not a mode of poisoning commonly witnessed in the human subject. It is almost entirely confined to those cases which arise from the bites of rabid animals or venomous serpents, or in which by any accident, noxious animal matter is introduced through a wound. In experiments on animals, it is occasionally resorted to for the purpose of illustrating the effects of such poisons as strychnia, morphia, arsenic, corrosive sublimate, and tartar emetic. The quantity of poison removed by absorption, has been determined by inclosing it in a small bag, introducing it into the wound, and noting the loss after given periods. The general conclusion from these experiments is, that but a small quantity of poison is required to destroy life, and that the effects of poisons are more rapidly manifested when they are brought into contact with the cellular membrane than under any other circumstances.

The absorption of poisons in the *stomach* is modified by the full or empty condition of this organ. The process is most rapid when the stomach is empty. Some poisons,—the woorara, and the poison of serpents, which are readily absorbed through a wound,—appear to resist the absorbing action of the stomach; and, according to Galtier (Toxicologie Générale, 1855, p. 8), this is not owing to any action of the gastric fluids or any change in the nature of the substance, because the poison when removed is still capable of acting as such by inoculation. The poison of glanders appears to resemble the poison of serpents (*echidnine*) in this respect. Professor Spooner states that he has administered the virus of glanders by the mouth without any injury whatever resulting.

But a point charged with this virus inserted into the flesh, is sufficient to propagate the disorder in all its virulence. (Med. Times and Gazette, April 11, 1857, p. 364.)

The mucous surface of the *small intestines* absorbs poisons with greater rapidity and uniformity than that of the stomach; and from experiments on dogs performed by Roselli and Gaetano Strombio, the same difference is observed with respect to the *rectum*. As arsenic, corrosive sublimate, laudanum, and other poisons, have of late years been criminally administered in several instances in the form of enemata, the results are of some interest. A quarter of a grain of strychnia dissolved in spirit was administered to dogs, in one set of cases by the stomach, and in the other by the rectum, care being taken that both were empty before the poison was introduced. The maximum period for the commencement of the symptoms by the *stomach* was in from thirteen to fifteen minutes, the minimum period from ten to twelve minutes;—while by the *rectum* the periods were respectively ten to twelve minutes and four to ten minutes. The period of death also differed: the dog which received the poison by the stomach died in sixty-five minutes, while that which had the poison by the rectum died in forty minutes. It was further noticed that while the sixteenth part of a grain of strychnia killed three dogs when administered by the rectum,—the tetanic spasms in two being very slight,—the same dose of this poison given by the stomach did not cause death. In respect to the salts of morphia, the symptoms by the stomach commenced in from three to nine minutes, whereas, by the rectum they appeared in from two to six minutes. (Galtier, op. cit. 9.) While these results possess a certain value in reference to the action of some poisons on the stomach and rectum, it is necessary to bear in mind that the period of commencement of symptoms and the period of death are variable in different animals.

The mucous membrane of the *lungs* by reason of its thinness, its great extent, and the absence of a protecting epithelial surface in the air-cells, is well-adapted for absorbing aerial poisons, and conveying them at once into the blood. The action of the vapors of ether and chloroform, as well as of the poisonous gases, sulphuretted hydrogen and carbonic acid, furnish instances of the rapidity and energy with which poisons produce their effects through the lungs. Substances which in large doses cause scarcely any noxious effects if introduced into the stomach and bowels, operate in small quantity and with great energy through the pulmonary membrane. Even when the quantity of poison is small, as where only a hundredth part of sulphuretted hydrogen or a tenth part of carbonic acid gas is diffused through the air, the noxious substance readily finds its way into the blood; and being absorbed and circulated with greater rapidity than it is eliminated, the blood is permanently poisoned and death is the result. The frequency with which respiration is performed compensates for the small proportion of the poison diffused through the air. It is through this medium that poisonous miasmata and the poisons of contagious diseases are received into the body. There is reason to believe that even mineral vapors, such as those of mercury, carbonate of lead, arsenious acid, and arsenite of copper, occasionally enter the blood through the lungs and produce the usual effect of chronic poisoning.

When a poison is introduced directly into the *blood*, it will be understood that its effects are rapidly produced. Dr. Christison found that when the muriate of conia was injected into the femoral vein of a dog, he was unable, with his watch in his hand, to notice any appreciable interval between the moment at which it was injected and that in which the dog died. The interval did not exceed three, or at most, four seconds. Prussic acid and strychnia act with great rapidity under these circumstances. Some poisons which appear to act readily by the blood, resist absorption by the mucous membrane of the alimentary canal (supra, p. 36). The poison of the viper (echidnine) possesses this property. It is fatal in small quantity when introduced into the blood through a wound, although it does not destroy the life of an animal when injected into the stomach. The poison of rabies is probably of this character; and there is no doubt that animal matter in a putrid state, which would operate as a poison if introduced into a wound, would be inert in the stomach or bowels. Bernard found in accordance with the statement of Galtier (ante, p. 37) that woorara destroyed the life of a bird in a few seconds, when a small quantity was injected into a wound; but when thrown into the stomach it had no effect, although on being removed from the stomach its poisonous properties were unchanged, and it was still found capable of producing death when injected into a wound. (Leçons sur les Effets des Substances Toxiques, 1857, pp. 61, 239.) It is necessary, however, that the animal should not be fasting, or the poison may be sufficiently absorbed by the mucous membrane of the stomach to cause death. (Op. cit. p. 291.)

Entrance into the *blood*, therefore, either by absorption or by injection, appears to be a condition necessary to the manifestation of the effects of the greater number of poisons. This was the doctrine long since taught by Magendie, and in more recent times it has received strong support from the experiments of Mr. Blake, M. Bernard, and others. These experimentalists have arrived at the conclusion, that not only are all poisons absorbed into the blood, but that absorption into the circulation is in every case a condition indispensable to their action. According to M. Bernard it is through the arterial capillary system of vessels that the effects of poison are first manifested, and until the substance has reached these vessels, however deadly it may be, there is no symptom indicative of poisoning. Strychnia and prussic acid applied directly to the brain produce no effect or only a slight local action after some time; but when a portion of either of these poisons is carried by absorption into the arterial capillary system, the symptoms of poisoning appear. (Leçons sur les Effets des Substances Toxiques, Paris, 1857, p. 47.) There are some objections to the universal application of this theory which will be a subject for consideration hereafter. (See post, Local Action of Poisons.)

Poisons in the Blood.—That a large number of substances comprising medicines and poisons enter into the blood and are thereby diffused over the whole of the body, has been clearly established by the discovery of them in that liquid, as well as in the secretions and excretions derived from it, and in the soft organs, such as the liver, spleen, heart, and muscular system. This diffusion of mineral substances by means of the circulation was in the first instance established by experiments on animals. In the year 1832, the late Mr. Aston Key introduced a quantity of ferrocyanide of potassium into a wound on the inside of the thigh of a donkey. In *six hours* afterwards the animal was killed, and he forwarded to me for analysis one portion of blood taken from the femoral vein, another portion from the mesenteric veins, and lastly, the contents of the thoracic duct. The ferrocyanide was readily detected in the three specimens, being most abundant in the blood of the femoral vein, and least abundant in the contents of the thoracic duct. In a set of experiments performed by Rapp of Tubingen with carbazotic or picric acid, a neurotic poison, the fact of the universal diffusion of the substance in the blood was made evident after death by the color imparted by the poison to the various textures and fluids of the body. In a fox killed in an hour and a half by swallowing sixteen grains of the acid, the white membrane of the eyes, the aqueous humor, the capsule of the lens, the membranes of the arteries, and in many places the cellular tissue, had acquired a lemon-yellow color. In other experiments the stomach was dyed yellow; there was a yellowness of the fibrine of the blood, and the urine was tinged yellow. Although this poison affects the brain and spinal cord, producing convulsions and insensibility, it is a remarkable fact that in no instance was there any yellow tint in these parts. (Christison on Poisons, 4th ed. 796.) Similar results as to the coloration of the organs have been since obtained by Mr. Calvert and Dr. Moffatt, of Manchester. They have further ascertained that the whole is eliminated in a few days chiefly by the urine. It has been ingeniously suggested that the addition of a small quantity of picric acid to colorless poisons would, by the yellow color imparted to the tissues, serve to indicate that poison had been taken. This species of evidence however in the absence of the poison itself would not be satisfactory. In persons poisoned by sulphate of indigo, the urine after the lapse of some hours has been observed to acquire a blue color. In some instances the *odor* of the poison clearly proves its diffusion. Prussic acid, the oil of bitter almonds, camphor, alcohol, chloroform, ether, oil of turpentine, and among the deadly agents, nicotine, have been perceived by the odor, not only in the stomach but in the brain and other parts of the body to which they must have been conveyed by the blood. In poisoning by phosphorus, the diffusion of the poison is traceable not only by its garlic odor, but by its property of luminosity under slow oxidation. The intestines and even the flesh of animals poisoned by phosphorus have been observed to emit the odor of garlic, and in the dark they have appeared luminous. In the case of a female who died while taking phosphorus medicinally, the whole of the viscera of the body were *luminous* in the dark. Facts of this description, by which the presence of poison is rendered evident to the senses, show even in a more satisfactory manner than chemical tests, that the soft parts of the body are universally penetrated by the substances.

In cases in which neither color nor odor will aid the inquirer, chemistry serves to reveal the presence of the poison. The important discovery first announced by Orfila in 1839—that arsenic could be detected and separated from the blood, secretions or viscera, of persons who had died from it effects, produced a complete revolution in this department of toxicology. It mattered not from what part of the body the blood was taken, arsenic was equally discovered; so that from these and other experiments, it appeared that the living or dead body in a case of arsenical poisoning, is for a time penetrated throughout by the poison, and that during life it was eliminated in the urine and other secretions. The fact that arsenic may be detected in the blood and urine of a person who survives its effects, is a point of considerable importance in a medico-legal view. Thus an analysis of either of these fluids may furnish evidence otherwise only satisfactorily obtained by an examination of the dead body; and cases of the criminal administration of arsenic to the living, which had hitherto escaped the hands of justice, owing to the absence of chemical proof, have thus become as clearly established to the satisfaction of a jury, as if the poison had operated fatally and had been found after death in the stomach. Arsenic has been in numerous instances discovered by toxicologists in the viscera of those who have been poisoned by it, even after the bodies had been interred for many years. Antimony was detected by Orfila in the urine of persons to whom tartar emetic was administered, and also in the substance of the viscera of animals killed by it. It was not discovered by him either in the blood or in any of the liquids of the body except the urine. It has since been found in both. Copper was detected by Orfila in the substance of the viscera of animals to which the poisonous salts of this metal had been given, but not in the blood or secretions. Tiedemann and Gmelin discovered verdigris (?) in the venous blood of horses poisoned by it; and the same chemists detected acetate of lead under similar circumstances. In an accident which occurred to a cow, where the animal swallowed a quantity of carbonate of lead mixed for paint, I detected traces of lead in the milk drawn some hours after the poison had been taken. (Guy's Hospital Reports, No. xii, 1841.) M. de Kramer, of Milan, detected nitrate of potash in the blood, urine, and fæces of persons to whom this salt was exhibited,—the iodide of potassium in the blood, chyle, and urine,—and iodine in the blood of a kid which had been made to respire the vapor of that substance. In other experiments, he found the chloride of barium,—tartar emetic, and nitrate of silver in the blood and fæces. (For the details, see Ann. d'Hyg. April, 1843, 415.) According to the researches of Dr. Percy it would appear that alcohol also enters into the blood, and is speedily conveyed to the brain, in which organ, as well as in the liver, he succeeded in detecting it by distillation. He also found it in the blood, bile, and urine. (Experimental Inquiry on Alcoholic Poisoning, &c., 1839, p. 59.) Lassaigne detected ether in the serum of venous blood of animals which had respired ether-vapor, and chloroform has been detected in the blood by Dr. Snow and others under similar circumstances. In fact all mineral and some vegetable poisons which are readily detected by chemical processes out of the body, have been found by different experimentalists in the blood, secretions, or soft organs of animals to which they had been administered. Cyanide of mercury and chloride of barium have been detected in the blood of the vena portæ (liver) and of the splenic vein (spleen) of the horse. Wöhler found in the urine of dogs and horses iodine, sulphuret of potassium, nitrate of potash, sulphocyanide of potassium, the salts of nickel, the oxalic, tartaric, citric, malic, gallic, succinic, and benzoic acids. Orfila has detected arsenious and arsenic acids—the arsenites—the soluble arseniates, tartarized antimony, iodine, potash, baryta and its salts, the mineral acids, sulphuric, nitric, and muriatic—ammonia, muriate of ammonia, and the soluble salts of copper, lead, mercury, gold, and silver. Whether the poison was introduced into the stomach, or applied externally, he equally detected it in the blood. (Toxicologie, i, p. 18.)

The facts above stated afford all the evidence that need be desired to prove that a large number of poisons enter the blood. In respect to those which have a local chemical action, this entrance into the circulation by absorption is a mere incident, and by no means necessary to their operation. The nitrate of silver is a corrosive poison, and proves fatal by producing an extensive destruction and disorganization of the viscera. Absorption in this case does not appear to be necessary to its poisonous action, yet it is undoubted that when this substance is exhibited in small doses for medicinal purposes, it is conveyed in some form into the circulation,—a fact established by the peculiar discoloration of the skin of the face and hands, produced by its long-continued employment in medicinal doses. It is impossible to say in what form it is transmitted, since, unless the ordinary chemical affinities are suspended by the powers of life, it is not easy to perceive how nitrate of silver could as such be circulated with the albumen or salts of the serum. Some other corrosive poisons, such as potash and the mineral acids, having a purely local action, are no doubt capable of entering into the circulation. With regard to potash, its chemical effects may be soon observed on the urine, although analysis may fail to detect it either in this fluid or in the blood. A case reported by Dr. Letheby, shows that even when sulphuric acid is taken in a concentrated form it may be absorbed, and eliminated in the urine. A boy, aged nine years, swallowed an ounce of the acid, and recovered in a few days. For the first four days, a large quantity of sulphuric acid was passed with the urine. It is stated that as much as one hundred and thirty-three grains of the strongest oil of vitriol were thus eliminated by the urine. (Med. Gaz. xxxix, 116.)

In a case of poisoning by this acid, which occurred to Dr. Geoghegan, of Dublin, a minute examination of the soft organs was made with a view of determining whether any of the poison remained in the tissues. This gentleman came to the conclusion that in cases of poisoning, the acid may be detected in the blood and viscera: the quantity relatively to the weight of structure operated on is greatest in the liver, and in proportion to that of the normal sulphates, in the blood. The tissues of the organs were decidedly acid to litmus. (Med. Gaz. xlviii, 331.)

Although the *alkaloidal poisons* are undoubtedly absorbed and produce their effects through absorption, chemistry has hitherto failed to detect and separate them from the blood, excretions, and tissues, with that rigorous certainty which a medico-legal investigation demands. In reference to one, *e. g.*, Atropia (from Belladonna), the presence of the poison in the blood and excretions has been inferred from the physiological action of its extract in dilating the pupil of the eye: but there are other poisons which produce the same effect. Some alkaloids (Daturia) have been pronounced to be present in the urine from crystalline form; others (Picrotoxia, Strychnia) have been inferred to be present in liquids by reason of the extreme bitterness of taste: and others again (Nicotine, Conia), by their very peculiar odor. Within a recent period many researches have been industriously made with reference to strychnia, and it has been very confidently announced that absorbed strychnia may always be detected by chemical tests in the blood, secretions, soft organs, and even in the bones. Without here anticipating that which will be more properly considered in another part of the volume, I may remark that absorbed arsenic and other metals may be procured in such quantity from the tissues as to allow of the application of various corroborative tests. In short, the demonstration of the presence of the poison is complete. The separation of absorbed strychnia from the blood and tissues has never, so far as I know, been thus demonstrated. The alkaloid has been pronounced to be present from the application of a single test, which is much the same in medico-legal practice as the affirming of the presence of arsenic from a deposit produced on copper, without verifying the nature of the deposit, by any corroborative processes.

Some experiments have shown that direct contact of the poison with the body is not absolutely necessary to the process of absorption. Imbibition and percolation through porous substances will equally allow of its penetration into the blood, although the effects may be more slowly manifested. Mr. Horsley, of Cheltenham, gave to a dog two grains of strychnia in a pill with conserve of roses wrapped in blotting paper. Three hours elapsed without any symptom of poisoning showing itself: in the morning, the dog was found dead. When the stomach was opened, the pill was found still inclosed in the paper wrapper, and on drying it, it was found to have lost only three-quarters of a grain. This had been removed by imbibition and absorption through the pores of the paper. Mr. Devonshire gave a grain of strychnia closely wrapped in paper to a cat. The animal died with the usual symptoms. The greater portion of the strychnia was found still wrapped in paper lying in the stomach of the cat. (See Guy's Hospital Reports, October, 1856, p. 336.) It must be obvious from these results that the portion of poison only which passes into the body by absorption destroys life, and that in reference to strychnia, this quantity is very small. The portion which remains in the stomach unabsorbed has no share in causing death; it is merely the surplus of the fatal dose whether it be wrapped in paper or lying in the stomach in a free state.

Poisons thus absorbed are diffused through the body and are either deposited in the organs, or slowly eliminated in the secretions if the individual should survive the effects. These agents appear to fix themselves more in certain organs and secretions than in others. Thus, for example, in cases of arsenical poisoning, the liver, probably from its containing an enormous quantity of blood, and from its proximity to the stomach, is generally more strongly impregnated with arsenic than the other soft organs. The proportion of absorbed arsenic found in it is, according to M. Flandin, nine-tenths of the whole quantity carried into the circulation. When arsenic is not found in the contents of the stomach, and death has taken place within the usual period, it may commonly be detected in the liver. In some cases of arsenical poisoning, I have not been able to procure any trace of arsenic from the stomach and intestines, or from the blood; but abundant evidence of the presence of the poison was obtained from an examination of the liver. It is important, therefore, in examining a body when death from arsenic is suspected, to remove for analysis the liver as well as the stomach.

Deposition of poisons in the tissues.—The deposition of poisons in the organs of the body, and their elimination by the secretions, have of late given rise to some important questions respecting the date of administration and the non-discovery of these agents by chemical processes. How long will a poison once deposited in an organ remain there? By what channel is it eliminated? When does this elimination commence, and when is it completed? The facts obtained from the human body and from experiments on animals show great differences among the different poisons. The functions of absorption and elimination are probably not the same in man and animals; and among human beings it may be considered that they are performed more rapidly in the child than in the adult, in the female than in the male, and in the healthy and vigorous, than in aged persons. The condition of the body must also affect these functions; and the effects produced by the poison itself must to a certain extent influence them.

That substances whether regarded as poisons or medicines, are rapidly removed from the body is a fact now well known to physiologists. This is especially observed with respect to those bodies that have no chemical action on the blood, or which do not form insoluble compounds with the tissues. The iodide of potassium taken into the stomach has been found in the urine in less than ten minutes. Its presence has also been proved in the saliva, milk, and perspiration. (Galtier, op. cit. 19.) In a case in which iodide of potassium was taken medicinally for fifty days, and the administration of it had been suspended for *seven days*, Kramer could not find a trace of it in the urine. It was inferred that it had been completely eliminated, an inference, however, which is scarcely justifiable, since recent researches show that a poison may cease to appear in the urine, and yet some portion of it may be retained by the organs. In one experiment in which ferrocyanide of potassium was given to a horse, the animal was killed in two hours, and the salt was found in the stomach, kidneys, bladder, as well as in the venous blood. In another horse killed after forty-eight hours, the whole of the salt had been eliminated by the urine; not a trace of it remained in the stomach or in any part of the body.

In a case of extroversion of the bladder, Mr. Erichsen observed that the ferrocyanide of potassium, taken in solution, was detected in the urine at different periods,—the earliest period being only *one minute* after it had been swallowed, and the longest *thirty-nine minutes*. An analysis of the cases showed that the time of its appearance in this secretion, depended upon the state of the stomach when the salt was swallowed: the earliest period being when the stomach was empty, and the latest when it was full, and the process of digestion was going on. Some vegetable salts and infusions were tried, and the only inference deducible was, that they required a much greater average time for their passage through the system, than the ferrocyanide of potassium. They gave some indications in the urine in from sixteen to thirty-five minutes. As to *elimination* by the urine, it was found that when the dose of ferrocyanide of potassium was forty grains, and its presence in the urine was manifested in two minutes, no trace of it could be detected after the lapse of twenty-four hours. Supposing the whole of it to have been eliminated by the urine, it must have escaped at an average rate of a grain in thirty-six seconds. It is rather remarkable that, when the dose was reduced by one-half, the salt was detected in the urine for an equal, and in one instance for a longer period of time (twenty-eight hours). Hence, the rate of elimination, like that of absorption, is by no means uniform. (For a full account of Mr. Erichsen's experiments, I must refer the reader to the Medical Gazette, xxxvi, 363, 410.) The results give a fair analogical explanation of the fact why poisons taken on an empty stomach operate with such great rapidity. The effect of animal poisons may be similarly influenced: thus the animal poison introduced into the system by dissection wounds, or by handling the viscera and liquids of a dead body when there is an abrasion on the fingers may, if the stomach be empty, lead to very rapid absorption and diffusion.

M. Audouard ascertained, in his experiments on rabbits, that when mineral poisons were administered, so as not to produce death rapidly, distinct traces of the substance might be detected in the organs of the fœtus; and that, when death has taken place with great rapidity, indications of the presence of poison may always be found in the placenta. A woman, during the last four months of her pregnancy, had been under treatment with iodide of potassium. The liquor amnii, collected during parturition, was clearly proved to contain iodine. (Comptes Rendus, 1845, i, 878.) This observation has been confirmed by the more recent experiments of M. Flandin. He found that by the use of mineral poisons he could induce abortion in rabbits; and the bodies of the young invariably yielded traces of the poison. He thinks also, that, in poisoning the blood through absorption, abortion may be induced, by the destruction of the fœtus in the living female through the medium of the poisoned liquid. Abortion was thus caused in animals in three different experiments, by the agency of morphia,—in two female rabbits, which took the poison with their food, and in a bitch, which received the poison by subcutaneous absorption. (Gaz. Méd. 31 Juillet, 1847, 620.) This throws a new light upon the subject of abortion. It has been commonly supposed that the shock to the system, and not the poisoning of the fœtus, was the primary cause which led to the expulsion of the contents of the uterus. The observations receive corroboration from those made by Dr. Beatty, that the ergot of rye, given in large doses to females during parturition, may, in some cases, exert a poisonous action on the fœtus, and lead to its expulsion dead. Dr. Percy observed in the effects produced on animals poisoned by alcohol, that, in the greater number of instances, an interval of a few minutes passed before a total loss of sensibility supervened. Hence he infers, that absorption is generally necessary for the action of alcohol. He has not found that the evacuation of the contents of the stomach by the stomach-pump removed the symptoms,—a fact in favor of its acting by absorption. The same has been observed with respect to arsenic; the symptoms have not abated, and persons have died after the poison had been completely removed from the stomach partly by vomiting, and partly by mechanical means. It is also worthy of remark, that arsenic, when in a state of solution, is very rapidly absorbed. The effects produced by absorbed arsenic are, in some respects, different from those excited by its local contact; and thus we may trace sometimes the exact period of its entrance into the circulation. Faintness, syncope, and general depression, with an indescribable uneasiness, are among the first symptoms caused by absorbed arsenic; and, in a series of cases which I had to examine, these symptoms showed themselves in from five to ten minutes after the poison had been taken in the state of solution. (See ARSENIC, post.)

Arsenic.—Observations in the human subject regarding the absorption, deposition, and elimination of this poison, are not very numerous; but experiments on animals show that in the short period of *one hour and a half* the poison may be most extensively diffused throughout the body. Orfila gave to a dog, while fasting, fifteen grains of arsenic dissolved in distilled water. The gullet was tied to prevent vomiting. About half a pound of blood gave a notable quantity of arsenic. The poison was also found in the liver, spleen, kidneys, heart, lungs, and brain, treated separately after washing from them the blood which adhered to them. There was barely a trace in the brain,—the lungs contained a small quantity,—the heart and kidneys more, and the liver and spleen yielded the largest quantity. The muscles and bones contained an appreciable quantity. When three grains were applied in a solid state to the cellular tissue on the back of a dog, the animal vomited in half an hour and died in *four hours*. On examination, the liver, spleen, kidneys, lungs, heart, brain, and alimentary canal (stomach and intestines), as well as the muscles, separately tested, yielded arsenic,—the largest proportion having been obtained from the liver, spleen, and kidneys (Toxicologie, ed. 5ème, 1852, i, pp. 381, 383).

The commencement of elimination and the period for the entire disappearance of arsenic from the body were determined by the following experiment. About two grains (1·8 gr.) of arsenic in fine powder were introduced into the thigh of a dog, and a few hours afterwards, medicines were thrown into the stomach to produce an action on the kidneys, so as to aid the elimination of the poison. In eight hours the animal, which had not vomited, passed a large quantity of urine. The urine was collected up to the fourth day, and each sample, when separately analyzed, yielded arsenic. On the twelfth day the animal ate its food readily, and appeared to have perfectly recovered. It was killed. The liver, kidneys, spleen, lungs, and heart, when examined by the same processes, did not yield the slightest trace of arsenic (op. cit. 384). The whole of the poison had thus been eliminated. From a table furnished by Orfila (op. cit. p. 433), it appears that arsenic was found in the urine of horses (passed while the animal was living), in from three hours and a half, to seven hours, after the administration of the poison. But it was found in the urine contained in the bladder of the dead horse within the short period of an hour after administration: in dogs it was found in urine passed, in from three hours and three-quarters to five hours after the injection of the arsenic.

From his experience and researches, Orfila arrived at the conclusion that *absorbed arsenic* does not remain for an indefinite period in the organs of the body, and that it is entirely eliminated in the human subject in from *twelve to fifteen days*. This is on the assumption that there is no suppression of urine, or of the other secretions. The elimination goes on to a certain extent by the intestinal canal, and by the skin. MM. Danger and Flandin think that this poison escapes from the system by the liver, as well as by the lungs and skin. (Traité des Poisons, i, 568.) I have frequently found it in very large proportion in the bile; and there can be no doubt that this secretion is an important channel of elimination. Dr. Geoghegan, in a valuable paper on Arsenical Poisoning, published in the Dublin Medical Journal, after remarking that the leading channel for the escape of this poison is the urine, relates the case of a child who recovered after swallowing a quantity of arsenic which had proved sufficient to kill a younger brother in nine hours. In this case no arsenic could be discovered in the small quantity of urine passed up to the fourteenth hour,—that of from fourteen to thirty-six hours, yielded faint and dubious indications,—in the secretion passed on the fifth day there was none; and none in the fæces passed at about thirty-six hours. In another case no trace of arsenic could be found in six ounces of urine passed on the sixth day (pp. 99, 115). In a case of poisoning by arseniuretted hydrogen which proved fatal in six days, Dr. O'Reilly found arsenic in ten ounces of a reddish serous liquid taken from the chest.

With regard to its deposition in, and removal from the liver, the various cases which were examined by Dr. Geoghegan, yielded the following results. Assuming the average weight of the human liver to be three and a half pounds, the total amount of arsenic deposited in this organ was:

After taking the poison.	Total weight of arsenic.
In 5½ to 7 hours,	0·8 grains.
8¾ "	1·2 "
15 "	2·0 "
17 to 20 "	1·3 "
10½ days,	1·5 "
14 days,	0·17 "

I have found arsenic in the liver in so short a period as *four hours*, and in another instance in six hours, the cases having proved fatal within these periods. The first of these was the case of a man named Burton, referred to me by Mr. Gell, coroner for Sussex, in May, 1854. The man died within four hours after he had been attacked with symptoms of poisoning by arsenic. Arsenic was found in small quantity in the stomach, duodenum, and rectum. It was also detected in the liver and spleen,—the latter organ containing a larger proportion than the former. In a medico-legal case which occurred to Mr. Chevallier, a man died from arsenic in *ten hours*, and the poison was found after death in the stomach, intestines, liver, and spleen. (Ann. d'Hyg. 1848, 1, 419.) Observations connected with the progressive disappearance of arsenic from the liver are still more limited. In a case of poisoning by arsenic (March, 1858), which proved fatal in twenty-six hours, the quantity taken being unknown—not more than a quarter of a grain was found in the stomach, and three-quarters of a grain in the intestines. It was estimated that not more than one grain and a quarter was present in the whole liver. The arsenic deposited in this organ had probably reached its maximum, and was disappearing, as it was freely contained in the bile. These facts are important in reference to the probable period of administration. On the other hand, in a protracted case of poisoning by arsenic, which proved fatal only after *seven* days, I did not find a trace of arsenic in the liver. (Guy's Hospital Reports, vol. vii, pt. 1.) Dr. Geoghegan's results lead to the conclusion that the liver acquires its maximum saturation in about *fifteen hours;* but, as he remarks, it is improbable that such quantitative results should be the same in different cases. They corroborate, however, those obtained by Orfila, in showing that on or about the fourteenth day, absorbed arsenic has either disappeared, or is rapidly disappearing from the liver, and it is worthy of remark, that at this date Dr. Geoghegan could not detect any arsenic in the tissues of the intestinal canal, kidneys, or lungs. In one case he found arsenic in the fæces on the fifth day, and in the contents of the colon on another occasion so late as the twelfth day.

Dr. Maclagan, of Edinburgh, met with a case in which a woman swallowed half a dessert-spoonful of arsenic, on the 4th of November, and was under treatment until the 29th, when she left the hospital, having recovered from the effects of the poison. There was, in the first instance, active vomiting; and on analysis it was found that the vomited matters contained arsenic. The urine was not examined until the second day (Nov. 6th). It then gave, by Marsh's process, an abundant arsenical deposit. On the fourth day, twelve ounces also gave a copious deposit of arsenic. On the fifth day, ten ounces gave a smaller quantity. On the ninth day, the poison was still found: on the fifteenth day, twenty-four ounces gave only a small quantity: on the twenty-first day, twenty-six ounces gave a faint deposit, and on the twenty-fifth day, not a trace could be detected. (Ed. Monthly Journal, vol. xiv, p. 131, 1852.)

It is important to observe that, as in the case of antimony, arsenic may still remain in the body, while it may not appear daily in the urine. In the case of the Duke de Praslin, who died six days after he had taken a large dose of arsenic, the poison was found in the liver and in the intestines, but none was found in ten ounces of the urine passed shortly before his death. (Ann. d'Hygiene, 1847, p. 402.) In giving arsenic to dogs for a period of nine months, in doses gradually increased, Danger and Flandin, state that they repeatedly analyzed the urine, without finding arsenic. (Op. cit. i, 737.) It was probably removed from the blood, and temporarily deposited in the soft organs. This deposition may be regarded as vicarious of elimination.

A case was communicated to the Pathological Society of London, in December, 1846, by Dr. Letheby, from which it appears that a young woman died in thirty-six hours after having swallowed, in two ounces of liquid, a dose of the poison equivalent to two grains and a half of arsenious acid. After death the arsenic (absorbed) was detected in the tissue of the stomach. (Med. Gaz. xxxix, p. 116.) As in this case the actual quantity of poison taken was known, as well as the period which the individual survived, it is obvious that, admitting the elimination of the poison not to commence for twelve hours, the quantity which passed off by all the secretions could not have amounted to the tenth part of a grain in an hour, or none would have been found in the tissues. From the experiments of M. Bonjean, of Chambery, it would appear that arsenic was detected in the urine of a patient who, one month before, had taken in twenty-four days only three-quarters of a grain of arseniate of soda! (Ann. d'Hyg. 1846, ii, 155.) The proportion thus eliminated may increase after the first day. MM. Danger and Flandin's experiments on sheep establish this so far as the urine is concerned; but in no instance did they find the quantity of poison thus passed to exceed the three-hundredths of a grain (·0308 gr.), even when the dose of arsenic was half an ounce. (Ann. d'Hyg. 1843, i, 472.) It appears, therefore, that the quantity of absorbed arsenic lost by elimination in the urine is sometimes very small.

MM. Danger and Flandin found that in sheep to which a large dose of arsenic (half an ounce) had been given, the poison first appeared in the urine and fæces in about *twenty-two hours;* that it was still discoverable in the urine fifteen days after it had ceased to appear in the fæces, and that it was altogether lost in the excretions, thirty-five days after the ingestion of the poison. When the animal was killed on the thirty-eighth day, not a trace of arsenic could be discovered in its body. (Ann. d'Hyg. 1843, p. 473.) It is difficult to infer, from the results obtained by such experiments, the period required for the entire elimination of the poison from the human body. Even in animals there is a difference. Thus, in young and vigorous dogs, arsenic was completely eliminated in from six to ten days, while in sheep the period varied from thirty to thirty-seven days, and the flesh was then safely employed as food. M. Flandin assigns, as an average, from one to two weeks for its complete disappearance. (Des Poisons, i, 738.) In other experiments it had entirely disappeared from the bodies of animals, *three days* after fifteen grains had been given (i, 737). There was no arsenic in the viscera, flesh, or bones, even where the dose had been gradually raised to this quantity. The form and dose in which the arsenic is administered, as well as the degree in which vomiting and purging have existed during life, must, among other circumstances, affect materially the duration of the period at which the poison will be detected, in the free or absorbed state, in the dead body. If the dose has been small, and the person has survived the effects for some days, the whole of the poison may have been expelled, and none found deposited in the soft organs, or in a free state in the stomach and intestines. In a case which occurred to me in November, 1846, a person died eight days after it was believed a very small dose of arsenic had been given to him. His body had been buried two years. It was disinterred; but, on examining most carefully the liver, stomach, pancreas, spleen, and a portion of the small intestines, not a particle of arsenic was found. The dose was small, the deceased had survived a long time, and there had been much vomiting while the deceased lived. Hence the circumstances were favorable for entire elimination. A similar fact was noticed in the cases of the Geerings of Guestling. A father and two sons died under symptoms of arsenical poisoning, at various periods. The bodies were exhumed; the appearances of poisoning were similar and well marked in the three cases. Arsenic was found in the bodies of the father and one of the sons; but there was no arsenic in the body of the other son. The stomach, intestines, liver, bile, and blood, did not yield a trace of the poison by processes of analysis, which readily demonstrated its presence in the other two cases. In the case in which it was not found, there was reason to believe, from the symptoms, that a dose of arsenic had been given a month before death; in the other cases, death had more speedily followed the administration of the poison, and it was probable that several doses had been given. (Reg. v. Geering, Sussex Summer Ass. 1849.) The wife, who was charged with the murder of her husband and two sons, was convicted of poisoning her husband, so that the absence of arsenic in one body, although made a subject of inquiry at the trial, was not material. The case of Dr. Alexander, of which the particulars were communicated to me by Dr. Geoghegan, is of great interest in reference to the elimination of arsenic. On the 16th of March, 1857, this gentleman took unknowingly a quantity of arsenic in arrow-root. Arsenic had been mixed with the arrow-root by mistake. The usual symptoms followed, and he died on the 1st April. The appearances were such as arsenic would produce: the stomach was ulcerated. Dr. Geoghegan made an analysis of the stomach and its contents, of the liver, spleen, and other viscera; but there was no arsenic in any of the organs, although the poison was abundantly contained in the arrow-root eaten by deceased. Thus, in *sixteen days*, the arsenic had been completely eliminated. This case further shows distinctly, in opposition to the rash and ignorant assertions of some medical witnesses, claiming to speak with authority, that a person may die from poison, and yet no trace of one of the most easily detectable poisons will be found in the body after death! (See Med. Times and Gazette, April 18, 1857, p. 388.)

In the case of Reg. v. Hunter (Liverpool Lent Ass. 1843), a medical witness was asked how long a period was required for the entire removal of arsenic (by absorption) from the body. There was reason to believe that the deceased had died from the effects of arsenic; but a difficulty in the case was, that although he had died within the short period of *three days* after the dose of arsenic could have been administered to him by the accused; and although the symptoms and the appearances in the body were such as might have been caused by arsenic, not a trace of that poison could be found in the stomach or bowels, or in their contents. The question, therefore, was, if this man had really died from arsenic, could every particle of the poison have been carried out of his body within the short period of three days? The analysis of the tissues was not so well understood then as it is now; but had these been examined, some absorbed arsenic might have been found, and the question thus practically solved. The medical witnesses answered the question by saying, that they thought the whole of the arsenic taken by deceased might have been removed from the body in *three days*, partly by vomiting, partly by purging, and partly by absorption, the poison being carried off by the urine and cutaneous exhalation. The answer was correct so far as it applied to their chemical examination; because they sought for the poison only in a free state in the contents of the stomach and bowels; and violent vomiting and purging might thus have got rid of a single dose taken three days previously; but it could not be applied with certainty to that portion of arsenic which is deposited in the liver and other organs. It is not probable, judging from ascertained facts, that the whole of the absorbed arsenic would have been removed from the liver and other soft parts, within so short a period as this; but, owing to the want of proof that there was poison in the body, the prisoner was acquitted.

In March, 1858, I examined, for Mr. Gell, coroner for Sussex, a case in which arsenic had been the cause of death in *twenty-six hours*, and the poison at this time had nearly disappeared from the parts of the body in which it is usually found. A man named Stephen Boys, after suffering from the usual symptoms as a result of taking some poisoned gruel, died within the period above mentioned. There was much vomiting and purging; the vomited matters could not be obtained for analysis, but a portion of the fæces yielded arsenic. The stomach and bowels were much inflamed; the inflammation was well marked in the duodenum and rectum. The gruel, of which the deceased had partaken freely, was procured, and apart from the quantity of poison deposited with the grits in the sediment, it contained in solution about forty-two grains of arsenic to a pint. The grits from which the gruel was made contained white arsenic in powder, in the proportion of sixty-four grains to an ounce. This, then, was a case in which, according to those who reason from ideas to facts, constituting a certain class of medical witnesses, a considerable quantity of arsenic should have been found in the body. The stomach and its contents, consisting of blood and mucus, yielded a quantity which could not be calculated at more than the eighth of a grain; in the spleen, pancreas, and duodenum, there was not a trace of arsenic. In three feet of the intestines, including the inflamed rectum, not more than three-quarters of a grain could be found; while the liver and gall-bladder did not contain, on the whole, more than one grain and a quarter. Four ounces of liver gave not more than one-fifteenth part of a grain; the bile yielded a larger proportion; hence it was a fair inference that the arsenic was in the act of being rapidly eliminated by this secretion. Altogether, the quantity found in this body, after a survivorship of twenty-six hours, amounted to two grains; and it is not improbable, that had the man survived five or six days, none would have been found.

The criminality of an accused person may actually rest upon an answer to this question, under another state of circumstances. In a case which occurred in France (July, 1844), the deceased, a man of the name of Lacoste, died, as it was alleged, from arsenic administered to him. It came out, in evidence, that for some time previously to his death, he had been in the habit of taking secretly a quack medicine, containing arsenic, for the purpose of curing himself of a disease of the skin of long standing. On an examination of the body, nine months after death, arsenic was found in the soft tissues, in which it had been deposited as a result of absorption. A person was charged with the murder of the deceased, and the medical question was, admitting that the deceased had died from arsenic, was this arsenic derived from the medicine taken voluntarily by him, or was it part of a dose criminally administered by the accused? It appeared distinctly in evidence that deceased had certainly taken none of the quack medicine for a period of *fifteen days* prior to his death; and it was contended for the prosecution, that no portion of *this* arsenic would have remained for so long a period in the body of a human being; therefore, it was argued, the arsenic found in the tissues must have been some portion of the poison administered subsequently, and that the prisoner was guilty of the crime. All the medical witnesses agreed that after *fourteen days*, no absorbed arsenic would probably be found in the tissues; that this would be the limit in the human subject, and, therefore, the arsenic found could not be ascribed to the medicine taken; that, in fact, this must have entirely disappeared during the interval of fifteen days. The point was considered too doubtful to rest upon presumption or conjecture, and the accused was acquitted. (See Flandin, Traité des Poisons, i, 687.)

The time allowed by the witnesses may be quite true of small doses of arsenic taken *medicinally;* but when the poison has been taken in powder, the time must be calculated from the date at which the last particle of solid arsenic remains in contact with the stomach or bowels, and so long as a person is living, there are no means of determining whether the unabsorbed poison is or is not entirely removed from these parts. So far, therefore, it is possible that the arsenic found in the body might have been due to the presence of that which had been voluntarily taken by the deceased. It appears that arsenic was found in small quantity only in the liver and intestines. Five ounces of liver yielded no more than the thirteenth part of a grain, and the intestines a still smaller quantity! Under these circumstances, as arsenic was not found in a free or unabsorbed state in the bowels, it was impossible to infer from the presence of so small a quantity, that it had been recently administered to deceased. It does not appear that the stomach or its contents were analyzed, or the discovery of arsenic in this organ, in a certain quantity, might have justified the medical inference that it had been recently administered. The facts proved were, however, consistent with the theory that the arsenic found was a portion of that which had been taken medicinally.

Arsenic, it is well known, may be deposited in the bones, and a question arose in one case whether it was deposited in the hair. A wealthy old lady died after a protracted illness ending in dropsy. A female relative, disappointed with the terms of her will, asserted that she had been murdered by the administration of small doses of arsenic over a long period. The woman went before the authorities, and stated that she had cut off some of the deceased's hair, while the body was lying in the coffin, that the hair had been analyzed by a chemist, and arsenic found in it. The case was subsequently submitted to Dr. Casper and

Prof. Mitscherlich, of Berlin, with the result that, on an accurate analysis of a portion of hair really cut from the head of deceased, not a trace of arsenic was found in it. The story told by the woman, who brought the charge of poisoning, was altogether improbable. There was no proof that the hair which she caused to be analyzed was taken from the head of the deceased, and she made no application for an analysis until after she had read in some medical book that the hair of animals, poisoned by arsenic, contained that mineral. There was also good reason to suspect that she had tampered with the hair. On the result of Mitscherlich's analysis, the complaint was dismissed as unfounded. (Casper Gericht. Med. i, 419, 1857.) There is no record of arsenic ever having been discovered in the hair of persons poisoned by this substance. I have examined the feathers of birds poisoned by arsenic, but none of the poison could be detected in them, although it was found in the bones, beak, and claws.

Although no definite conclusions can be drawn regarding the period and relative amount of deposition in the soft organs, or the period at which absorbed arsenic is entirely discharged from the body, there are certain leading points which are undisputed. In the first place, arsenic is not a normal constituent of the human body; this has been clearly proved by Orfila, under the eyes of a committee of scientific men. (Rapport sur les Moyens de constater la présence de l'Arsenic dans les Empoisonnemens par ce toxique, par M. Orfila, Paris, 1841.) Secondly, when introduced as a poison, it is absorbed, and although temporarily deposited in some of the organs, it is sooner or later eliminated, and the whole is removed from the body. The statement that when once deposited, it may remain for an indefinite period, has no foundation in fact. In recent cases of administration it may be found in the stomach and bowels, and not in the liver or other organs, while in cases of older date it may be found in the liver, after it has disappeared entirely from the stomach. Thus, in the case of the Atlee family, referred to me by Mr. Carter, coroner for Surrey, in January, 1854, the body of the woman was exhumed after a month's burial. Arsenic was not found in the *stomach* or *bowels*, but it was readily detected in a small portion of the *liver*. The poison had probably been taken several days before death. A similar case occurred to Dr. Geoghegan, and is reported by him in the paper referred to in the Dublin Medical Journal. (Case 1.)

The cases above related are of considerable importance in a medico-legal view. The conclusions which may be drawn from them are—1. That arsenic does not penetrate the liver by imbibition from the stomach after death. 2. That the mucous surface of the stomach is not a medium of elimination for arsenic deposited in the liver. 3. That no medical opinion of the absence of arsenic in a dead body can be expressed from a chemical examination of the stomach only. The liver and other viscera should be examined before the presence of the poison is negatived. 4. That absorbed arsenic is not necessarily found deposited in all the organs and tissues: it may be present in the liver and absent in the spleen and pancreas. 5. That a person may die from the effects of arsenic, and none of the poison may remain in the body. 6. That the quantity of arsenic remaining in the body of a person who has died from it may be small or large, according to circumstances which vary with every case. 7. That there may not be a sufficient quantity remaining to kill another person, although, making due allowance for the portion ejected, there may have been sufficient to destroy the person in whose body the poison is found.

It need hardly be observed, that when the criminality of a person is made to rest upon an answer to a medical question, in reference to the date of administration, a witness must be cautious in drawing conclusions. Thus, from the quantity of arsenic found in the liver it may be inquired whether this indicates or enables him to fix a particular date. The case of Ann Merritt, tried at the Central Criminal Court in March, 1850, presents a remarkable instance of the kind of medical evidence which may temporarily satisfy a court of law on questions of this magnitude. The accused was charged with having caused the death of her husband by arsenic. There was no doubt the deceased had taken arsenic and that he had died from its effects; but the question was, whether he had taken it by mistake for a soda-powder in the morning, or his wife had administered the poison to him in a dissolved form in some gruel which she had prepared later in the day.

The deceased was seen at his breakfast at eight o'clock in the morning; he was then sick: at twelve o'clock he was vomiting; and at five or six o'clock he was ill in bed, and complained of sickness and cramp in his feet. He died at half-past twelve the same night; the duration of his illness, from the time at which he first complained of sickness, having been rather less than seventeen hours. At the trial, Dr. Letheby stated that he had discovered eight grains and a half of arsenic dissolved in the fluid contents of the stomach, and a quantity, calculated at *two grains*, in the whole of the liver. In reply to questions respecting the time at which the arsenic had been taken, the witness said: "Looking at the quantity I found, and the parts I found it in, in my judgment the arsenic I found had been taken not more than *two or three hours before death*." . . . "My observation in reference to the *time* it had been taken, has reference both to the *stomach* and *liver*." . . . . *Question*. "Are the data at all safe?" *A*. "Yes. I will tell you why; I found in the stomach eight and a half grains of arsenic, and there was not much in the intestines. I conclude, therefore, there had not been time for it to have passed into the intestines, which would have been the case if it had been taken a long time before death: but there was only a trace in the intestines, so I conclude that it was taken a *very short time* before death." (Sessions Reports of Central Criminal Court, March, 1850, and London Medical Gazette, vol. xlvi, p. 291, August, 16, 1850.)

As there was no reason to suppose that deceased had taken arsenic for the purposes of suicide, and no one but the wife could have administered the poison to him, at the time so positively fixed by Dr. Letheby, —this was substantially removing all possibility of accident, and charging the woman with the murder of her husband. The prisoner was convicted and sentenced to death; but as the medical data relied on did not justify the strong inculpatory opinion which the witness had expressed, the sentence was commuted. Sir B. Brodie, Dr. Billing, Dr.

Leeson, and others interposed and satisfied the authorities that the statement respecting the time of administration was not in accordance with sound medical views. Instead of making a candid admission of the mistake, Dr. Letheby addressed a letter to the public journals charging these gentlemen with inexperience, and affirming that his opinions "were founded on fact, and that his evidence was the expression of truth."

This case has been frequently made a subject of quotation and comment. It holds out a warning to medical witnesses not hastily to deal with matters beyond the true scope of medical jurisprudence. There was not a single fact, chemical or physiological, to justify the fixing of the time at which arsenic was taken to two, three, or even four hours before death! There are very few cases on record in which arsenic has proved so rapidly fatal, and the amount of arsenic deposited in the liver in these cases has not been ascertained. Medical experience, however, so far as it goes, shows that it requires a much longer period than the time assigned, in order that two grains of arsenic should be found deposited in this organ. (Cases by Dr. Geoghegan, ante, page 46; also case of Boys at p. 50.) Dr. Geoghegan's researches lead to the conclusion that this quantity would not be found in the liver in less than fifteen hours after the poison had been taken. It was therefore probable that the deceased had taken the arsenic many hours before death, and there was no evidence to prove that he had had more than one dose. That eight grains of arsenic were found after death in the stomach, mixed with or even dissolved in gruel, proved nothing; for it is obvious that if the stomach of a person already contained arsenic, a large quantity of liquid taken subsequently might acquire a poisonous impregnation. The presence of this arsenic in the stomach, and of a much smaller quantity in the intestines, did not justify the opinion expressed, since it was impossible to say how much had been originally taken into the stomach, and how much had already passed through the bowels. The medical facts showed that the deceased had taken arsenic, and had died from the effects; but they did not indicate the quantity taken, or the time at which it had entered the body. There was evidence from symptoms that arsenic had probably been taken early in the day, and while the quantity assumed to exist in the liver was consistent with this supposition, it was inconsistent with the opinion given at the trial. Instead of being the expression of truth, the opinion, it will be perceived, was purely speculative, and wholly indefensible. This kind of evidence brings disgrace on legal medicine. It tends to the acquittal of the guilty, and the conviction of the innocent. In short, this very case was brought before the House of Commons as a strong argument for the abolition of the punishment of death for murder! It was argued, that the law should not inflict "an irrevocable punishment depending on medical opinions liable to dispute, and too much and unavoidable uncertainty." (Medical Gazette, vol. xlvi, pp. 213, 291.) This argument would have some force if these speculations fairly represented the general character of medical evidence.

In a case of poisoning by arsenic, which occurred in France some years since, the analysis of the liver induced MM. Bayard and Chevallier, two distinguished French medical jurists, to draw the conclusion, that arsenic had been taken only a few hours before death, because the liver *did not contain a trace of poison.* (Annales d'Hygiène et de Medecine Legale, vol. xxxv, i, 1846, page 149.) This is undoubtedly more in accordance with observation and experience regarding the absorption and deposition of arsenic. Its absence from the liver shows that it has been either too short a time in the body to be deposited in that organ, or that the poison has been taken for a sufficiently long period to have been entirely eliminated. (See case of Dr. Alexander, ante, p. 49.)

With reference to absorbed arsenic, there are two points requiring notice: 1. The extent to which it is diffused through the body; and 2. the absolute quantity deposited in the organs. In chronic poisoning, arising from the administration of small doses at intervals, I have found the arsenic extensively diffused, but in small proportion. The quantity deposited appears to depend on the largeness of the dose, or on the frequency with which small doses are repeated. There are some facts on record which show to how great an extent arsenic may be diffused when it has once entered the blood. In the case of a pregnant female poisoned by arsenic in the fourth month of pregnancy, the poison was detected by MM. Mareska and Lardos, in the body of the fœtus. It was also discovered in the uterus and placenta, the latter organ containing a larger proportion than the fœtus, but there was none in the liquor amnii. (Gaz. des Hôpitaux, Janvier, 1846.) Even the entozoa found in the human body become under these circumstances thoroughly impregnated with the poison. In case of a female poisoned by arsenic, whose viscera were forwarded to me for examination in July, 1845, I found the poison in a worm (*lumbricus*) which was discovered dead in the small intestines. (Guy's Hosp. Rep. October, 1846, p. 462.) In March, 1857, I was engaged in examining some fowls which had died from the effects of arsenic. In the crop and gizzard of a fine cock about twenty grains of the poison were found; and the whole body was thoroughly saturated with absorbed arsenic. It was separated from the blood, the liver, the muscles of the breast, the comb, the claws, and even the thigh-bones. In examining the body of a hen, the arsenic was found in large quantity in the ova contained in the oviduct, and particularly in the yolks of those which were developed.

It is proper to state, however, that these results are subject to exceptions, the cause of which is not well understood. Arsenic is not always found in the *blood*, although it may be found deposited in the soft organs. I have had occasion to make this observation in a few instances, and in a case reported by Dr. Geoghegan (No. ix, op. cit. p. 93), there was no arsenic in the blood, while the liver yielded a small quantity. Again, in a case fatal in nine hours, he discovered no arsenic in the muscular structure; while in another case, fatal in seven hours, he discovered it without difficulty in the proportion of one-thirteenth of a grain to the pound (op. cit. p. 113). M. Benoist, of Amiens, was required to make an official examination of the body of a girl who died in the sixth month of her pregnancy. It was alleged that she had committed suicide by taking a large dose of arsenic. The poison was clearly found in a solid state in the stomach. The fœtus was examined with a view of determining whether it had died from the effects of the poison. There was not the slightest trace of arsenic in the body. (Gazette des Hôpitaux, October 31, 1846, p. 512.) Such facts as these, ascertained with regard to a poison better known and understood by toxicologists than any other, show the extreme danger of laying down rules respecting the invariable retention in the body of other poisons on which experience is far more limited.

Antimony.—Some recent cases of poisoning have given an especial interest to questions connected with the absorption, deposition, and elimination of this metal. The only compound which requires consideration in this respect is tartar emetic or tartarized antimony. The fact, that antimony was absorbed into the blood was determined by Magendie in a set of physiological experiments in the year 1813, but the metal was not chemically detected in the blood and viscera until Orfila's researches on this subject in the year 1839. (Mémoires de l'Academie de Médecine, tome viii, p.509, March 10, 1840. See also Annales d'Hygiène, 1840, i, p. 474.) Tartarized antimony, in the dose of from fifteen to twenty-five grains dissolved in water, was introduced into the stomachs of dogs, and the gullets were tied to prevent vomiting. The animals died in a *few hours*. The liver and kidneys yielded a comparatively large quantity, the spleen, lungs, and heart contained barely traces of antimony. In an experiment in which thirty-one grains, in powder, were introduced into the wound in the thigh of a dog, the animal died in *twenty hours*. The lungs, heart, and spleen yielded not the least trace of antimony, and the liver gave only a small deposit. Two ounces of urine contained in the bladder yielded a large quantity.

In other experiments, in which the powder was applied to wounds in quantities varying from two grains to six, the following results were obtained. In one dog, destroyed *one hour* after the application, there was no antimony in six ounces of blood, and only a small quantity in the liver; in another, destroyed in *four hours*, six ounces of blood taken from the aorta, and six ounces taken from the vena cava, gave respectively no indications of antimony. The liver gave a large number of antimonial deposits, and the urine from the bladder also gave numerous deposits. In a third experiment, the dog died in *seventeen hours*,—the liver gave only traces of the metal, while the urine yielded numerous large deposits. Two grains of tartarized antimony had been applied to a wound in this case. In a fourth experiment, the same dose was applied, and the animal died in *thirty-six hours*. The liver yielded no antimony; the urine contained it abundantly. From these results, Orfila drew the conclusion that antimony was rapidly absorbed and as rapidly carried out of the body by the urine, so that after ten, twelve or fifteen days, none would be found in the liver and kidneys. MM. Millon and Laveran proceeded differently and obtained different results. 1. A dog was fed for ten days with food containing a daily dose of four and a half grains of tartarized antimony, making in the whole forty-five grains. The dog died *six days* after ceasing to take the antimony in its food, and obviously from the effects of that substance. The metal was found in the liver, muscles, coats of the intestines, the lungs, and the brain.

2. A second dog, similarly treated, died *thirteen days* after the withdrawal of the antimony. The metal was found in the various organs, but the brain appeared to be the most strongly impregnated with it. 3. A third dog, which recovered from the antimonial treatment, died suddenly *six weeks* after its cessation. The antimony was found in appreciable proportion in the liver and fat, but it had especially accumulated in the *bones*, i. e., in a tissue in which its presence is quite compatible with the healthy exercise of the bodily functions. 4. A fourth dog was killed *three months and a half* after the withdrawal of the antimony. On analysis the metal was chiefly found in the fat. The liver contained a small quantity, as well as the bones and other tissues; but the fat contained ten times the proportion found in all the other parts. 5. In this experiment, the dog was killed *four months* after the antimony had been withdrawn. The metal was found to be chiefly accumulated in the bones. The liver also contained a great deal: the other tissues contained very little. 6. A young bitch took tartarized antimony for five days, about *fifteen days* before parturition. The animals were killed and the livers of the pups yielded antimony. (Comptes Rendus, 1846, i, p. 1043. Orfila, Toxicologie, 1852, i, 628. Bouchardat, Annuaire de Therapeutique, 1847, p. 134.) Dr. Nevin has experimented on rabbits with the following results. The antimony was given, in doses varying from half a grain to one or two grains, over a period of several days: the total quantity given varying from twelve to seventy-two grains. The absorbed metal, like arsenic, was found by chemical analysis to be widely diffused through the body. It was most abundant in the liver and the kidneys. In the blood it was sparingly found at any time, and in the muscles only a trace, after the longest continued administration. It was also found in the livers and kidneys of the immature fœtuses of one rabbit, as well as in the placentas. It appeared to exert a fatal influence on the fœtuses in utero. 1. A rabbit, which had taken in divided doses four grains during twenty-four hours, was killed *four hours* after the last dose. Antimony was found in the stomach, but not in the large and small intestines. The liver contained a faint trace: but there was none in the blood or in any of the other parts examined. 2. Five grains were given in three days, and the animal was killed *four hours* after the last dose. Some antimony was found in the stomach and large intestines, a trace in the kidneys, a copious deposit was procured from the liver, and a well-marked deposit from the blood. 3. A rabbit died, poisoned by twelve grains, about *sixteen hours* after the last dose. Antimony was detected in small quantity in the stomach, intestines, kidneys, lungs, and blood: it was most abundant in the liver and in the urine. Exp. 7. This animal died poisoned *seven hours* after the last dose, the whole quantity administered having been fifty-one grains, given in *fifteen days*. Antimony was found in the large and small intestines —copiously in the liver, in the spleen, and urine, but in small quantity in the kidneys and lungs. 9. This animal was killed *fifteen days* after the last dose,—twenty-three grains having been given during a period of fifteen days. No antimony was found in the stomach and intestines: there was not a trace in the liver, but the metal was found in the bones. 10. Thirty grains were given over a similar period, and the rabbit was

killed *fifteen days* after the last dose. A trace of antimony was found in the stomach, cœcum, kidneys, and lungs,—none in the liver: the presence of the metal was very decided in the bones, urine, and excrement. 11. In this experiment fifty-one grains were given in fifteen days, and the animal was killed *one month* after the last dose. There was no antimony in the stomach: only a slight trace in the liver and lungs,—more in the kidneys. The largest proportion was in the bones. Twenty-one days after the last dose, the urine and excrement gave decided traces of antimony. (Liverpool Medical Journal, 1857, No. 1, p. 46, et seq.) From these experiments it appears that antimony was found in the bones on the fifteenth day after the last administration, and was still present on the thirty-first day in another experiment.

Dr. Mayerhofer, of Munich, gave to a healthy dog fifteen grains of tartarized antimony, dissolved. Seven hours afterwards, six grains in solution were rubbed on the thighs and abdomen. The animal died in fourteen hours; and shortly before death two grains were given by the mouth. On analysis, antimony was found in the stomach in a soluble form, probably from the recent administration. The metal was also detected in the blood of the heart, of the portal vein, in the liver, lungs, brain, intestinal canal, as well as in the urine passed during life. (Heller's Archiv. 1846, 111.) To some sheep affected with pneumonia one drachm of tartarized antimony was given medicinally (March, 1857). They were killed within about twelve hours of taking the medicine; and on analysis I found in the fleshy parts of the legs of the animals a considerable deposit of antimony.

This metal appears to be eliminated through the *milk*. A writer in the Medical Times and Gazette (May 23, 1857, p. 517) states that he administered to a cat which had given birth to five kittens, one-third of a grain of tartarized antimony twice, at an interval of eight hours. These doses caused vomiting, and so much prostration, that the course was changed to one-twelfth of a grain, which dose was repeated twice daily. Three days after this, one of the kittens was drowned, and its viscera subjected to analysis with the result of detecting distinct evidence of the poison. Three days later, the mother still receiving the poison, two of the other kittens were killed. In these, the poison was detected in the heart, including its contained blood and lungs taken together,—in the liver and kidneys taken together, and in the stomach and intestinal canal with their contents.

From the experiments of Dr. Brinton, of King's College, it appears that in certain cases in which antimony has not been introduced into the stomach, it is eliminated from the system by the mucous membrane of this organ. He injected into the superficial femoral vein of a large dog, ten grains of tartarized antimony dissolved in four ounces of water. The animal instantly fell into a state of collapse, without vomiting or purging. At the end of fifteen minutes it was killed, and the contents of its stomach, then in the act of digestion, were collected. They were found to contain tartarized antimony in large quantity (the quantity not stated). Dr. Brinton thought that the proportion present in the gastric fluids exceeded that in which it was mixed with the blood of the animal: in short that the poison was not only transferred from the thigh to the stomach, but that it was concentrated as well as eliminated in this latter cavity. (Medical Society of London; Lancet, May 31, 1856, p. 591.) It is unfortunate that this chemical question was not determined by experiment. As the quantity injected into the blood was accurately known, it should have been ascertained what proportion of antimony was contained in the gastric fluid as well as in the fluids of the intestines, how much remained in the blood, and how much had been deposited in the liver and other organs. The result, as it stands, simply shows that some portion of antimony is eliminated by the stomach within the short space of *fifteen minutes* after it has been injected into the blood of the femoral vein. Orfila ascertained with respect to arsenic, that the alimentary canal contained a portion of the poison in a dog killed in four hours by three grains applied in a solid state to the cellular tissue. It has been supposed that these results are adverse to any conclusion respecting recent administration, when antimony is found in the contents of the stomach: but this objection could only arise in those cases in which the person alleged to have been poisoned by antimony, had received the poison by injection into the blood—or by direct application to a wound or ulcer. Such a case as this has, I believe, never yet presented itself on a charge of poisoning; the poison has always found its way into the body either by the stomach or by the rectum, and the question therefore practically resolves itself into this, How long can tartarized antimony when swallowed, remain in the stomach? The answer must depend on the circumstances proved in each case. I am not aware of any facts showing that the stomach is a medium of elimination for antimony, when the metal has been once removed from it and deposited in the liver and other organs as a result of absorption originally from the stomach. It would be necessary to assume that the stomach and its contents had first been entirely cleared of every trace of the poison, and that the poison subsequently had returned to this organ from the parts to which it had been conveyed, and was again deposited in the contents! Adversely to this assumption, however, it has been ascertained that the stomach has frequently contained no trace of antimony after death, while the poison has been found rather strongly deposited in the liver and other parts. This negatives the assumption that when the stomach is once cleared of the poison, its mucous surface becomes in the *human body* a medium of elimination.

It is a remarkable fact, in reference to this question, that in instances of poisoning by antimony, in which there has been an opportunity of making an analysis, this metal has *not* been found in the stomach, whilst it has been found abundantly in the liver, spleen, and kidneys. Some cases elsewhere published (Guy's Hosp. Reports, Oct. 1857, Nos. 29, 30, and 32, p. 439), show that this theory, as applied to the human subject, is erroneous. Two children were poisoned, each by ten grains, and a third child by fifteen grains of tartarized antimony. They died respectively in eight, thirteen, and six hours. No antimony was found in the stomach or intestines. It cannot be doubted, as they died from the effects of antimonial poisoning, that some portion had been absorbed and deposited in the tissues of the organs. The absence of antimony from the alimentary canal is a clear proof that the mucous surface is not an eliminating medium, or some antimony would have there been found. In the case of McMullen (Liverpool Summer Assizes, 1856), it was distinctly proved that the deceased had died from the effects of tartarized antimony. Mr. Watson, of Bolton, made the analysis, and found no antimony in the stomach or contents, while he found it most abundantly in the liver, spleen, and kidneys. Such a result is inconsistent with the theory that the stomach is an eliminating surface for other organs. In fact, if that were so, as long as there was any antimony in the body it ought to be found in the stomach. When human beings are poisoned by injecting tartarized antimony into veins, or thrusting the powder into wounds, the result may be different; but experiments so conducted are not applicable to ordinary cases of poisoning in the human subject. As evidence of recent administration, less reliance can be placed on the discovery of antimony in the intestines. Antimony is eliminated in the bile, and as the liquid traverses the whole of the intestines, the metal found may have been derived from this secretion. If, however, the poison be present in the intestines, in proportionally large quantity,—if it be in a solid form,—if there be no poison in the bile taken from the gall-bladder, if the poison in the intestines be mixed with blood, mucus, or food, and not with bile, then this theory would not explain its presence in the intestines. Assuming that the intestinal mucus itself may become in certain cases a medium for the elimination of the poison from the body, some of the above-mentioned conditions might exist which would be inconsistent even with this assumption.

Observations regarding the absorption and elimination of antimony in the human body are at all times more valuable than those derived from experiments on animals, but unfortunately these are few in number, and the facts as yet known scarcely admit of a satisfactory generalization. Their importance, however, cannot be over-estimated. We are indebted to Orfila for the following observations. 1. In a patient affected with pneumonia, eighteen grains of tartarized antimony were given in twenty-four hours. Four ounces of urine, collected during this period, yielded metallic antimony. Much urine was lost, and there had been much purging. 2. In another case, nine grains were taken, dissolved, during the same period. About five ounces of urine (passed twelve hours after the last dose) yielded as much antimony as case 1. 3. The urine of four patients affected with pneumonia, to whom from twelve to eighteen grains of tartarized antimony had been given during twenty-four hours, yielded antimony; but the same process did not reveal the presence of any antimony in one patient who had taken only nine grains during that period. Some of these patients had had alvine evacuations. Martin Solon detected antimony in the urine of a patient who had taken only four grains of tartarized antimony, and who had had neither vomiting nor purging. 4. Tartarized antimony was prescribed in large doses for a patient. The urine collected *three days* after the last dose had been taken, yielded no antimony. 5. Antimony was found deposited in the liver, spleen, and kidneys of a patient who died *fifteen hours* after she had taken about eight grains of tartar emetic. This had caused some purging, but no vomiting. (Memoirs of the Academy of Medicine, April 7, 1840, p. 517.)

From the researches of MM. Millon and Laveran, it appears that there may be intermissions in the elimination of antimony. In giving to their patients from one grain and a half to five grains of tartarized antimony, they remarked that it was eliminated by the urinary secretion, but in some instances slowly and irregularly. They therefore examined the urine, not only several days after the introduction of the medicine, but for some days after it had ceased to appear in this secretion. They then found that its elimination underwent a well-marked intermission, and that, in a most unexpected manner, it appeared to remain for a certain period fixed in the body. In two patients they detected traces of it twenty-four days after its administration. In the body of one who died of phthisis, they found antimony in the liver. In a third case, antimony was detected in the urine after twenty days; in two others, after nineteen days; and in three others, after sixteen, seventeen, and eighteen days respectively. (Comptes Rendus, 1845, ii, 638.) The fact of the *intermittent elimination* of antimony is of importance, as it tends to show that even when traces of poison cease to appear in the secretions, it does not in all instances follow that the substance is entirely expelled from the body.

Among medico-legal cases which may be cited in illustration are the following. A woman, æt. 41, died under suspicious circumstances in February, 1845. About a week before death, some doses of tartarized antimony (some centigrammes, a c.-gr.=0·154 gr.) had been prescribed for her by her medical attendants. The body was exhumed and examined on the 18th of February. Traces of arsenic were found in the contents of the stomach, and of arsenic and antimony in the stomach and bowels; but no trace either of arsenic or antimony in the liver, lungs, and blood. (Ann. d'Hyg. 1846, i, 155.)

A patient of M. Marchal's, in a Parisian hospital, was treated with tartarized antimony in large doses. He died eight days after the cessation of the antimony. The result of a chemical examination was that the liver contained a considerable quantity of antimony,—that the kidneys contained a smaller quantity,—that the blood also yielded traces of the metal, while the brain contained much less than the other organs and the blood. (Journal de Chimie Médicale, 1853, p. 358.)

A child, eighteen months old, died twelve days after certain medicines, including tartarized antimony, had been given to it. As there was some suspicion that death had been caused by mineral poison, three-fourths of the intestines were minutely examined, but not a trace of antimony was found therein. (Casper's Leichen-Oeffnungen, 1853, part 2, page 156.) The case of a man named McMullen (Liverpool Autumn Assizes, 1856), who died from the effects of small doses of tartarized antimony administered at intervals during a period of four months, presents us with the results of an analysis *four days* after the withdrawal of the antimony. The metal was most abundantly found in the liver, spleen, kidneys, urine, and fæces. The quantity was very small in the heart, lungs, and rectum. It was *not present* in the stomach or its contents. There were traces in the blood. The other parts of the body were not examined for it. In a case reported by Dr. Haldane, in which a man, laboring under pneumonia, had taken from forty to fifty grains of tartarized antimony, death took place on the fourth day. A considerable quantity of antimony was found in the liver, and there was antimony in the affected lung, but less in the liver. (Edinburgh Monthly Journal, August, 1854, p. 184.)

Among cases which have occurred to myself, I have found traces of antimony in the liver when death had taken place in about fourteen hours after a dose of three grains of antimonial powder had been taken. (Guy's Hosp. Reports, vol. vii, part 1, case xix, May, 1850.) In February, 1853, a case was referred to me by Mr. Wakley, in which a man died twenty-four hours after he had taken three grains of tartarized antimony in solution. No trace of antimony was found in the stomach, intestines, or their contents. In 1856,I had an opportunity of making a complete examination of the whole of the viscera of a man who, five weeks before his death, had taken a solution of tartarized antimony in ordinary medicinal doses. No part of the viscera gave the slightest trace of antimony. In 1837 a similar investigation was made of the body of a female who had taken within three days of her death four grains of James's (antimonial) powder, and two grains of tartarized antimony in solution. Antimony was found in the small and large intestines; but there was no satisfactory evidence of it in the liver or other viscera. In a case on which I was consulted in March, 1857, a gentleman died two hours after three-quarters of a grain of tartar-emetic had been administered to him in solution. The viscera, generally, were carefully examined by MM. Tardieu and Lassaigne, with the result that they found therein no trace of antimony or of any other mineral poison. In April, 1857, a physician, in this metropolis, died under somewhat suspicious circumstances. In about two hours after his dinner he was seized with distension, pain in the abdomen, and vomiting. The bowels were constipated; the vomiting and pain continued throughout his illness, which terminated fatally on the fourteenth day. The stomach was so irritable that nothing could be retained on it. The vomited matters were of a yellow or greenish color. After death, the intestines were found highly inflamed, externally and internally. It was suspected that poison might have been administered to him on the day of his illness. The viscera were brought to me for analysis; antimony was found in the stomach and intestines, but there was no trace of it in one of the kidneys; and Dr. Marcet, who examined the liver, detected no trace of it in that organ. It was found on inquiry, that three grains of tartarized antimony, in powder, were given to the deceased six days before he died. Thus, then, in this instance, the antimonial medicine had not entirely quitted the alimentary canal. It was impossible to ascribe its presence here to elimination from the mucous surface, because there was no evidence of any deposit in those organs in which it is commonly found. The symptoms from which deceased suffered, and the appearances, taken as a whole, were unlike those of antimonial poisoning, and the presence of the metal was satisfactorily accounted for by its having been prescribed medicinally.

Taking these results generally, it would appear that antimony given in a large dose, or repeatedly in small doses, is rapidly absorbed and eliminated, chiefly by the urine. It is, at the same time, deposited in greater or less quantity in the tissues and organs. Under recent administration, if present in the body in sufficient quantity, it may be found in the stomach and bowels, and little or none may be present in the liver; after a variable time it disappears from the stomach and bowels, although it may be present in the fæces, while the liver, kidneys, and spleen may contain it in large, and the blood and other organs, in small proportion. That some weeks, or even months, after its introduction (in animals) the metal, if not entirely eliminated, will be found chiefly deposited in the fat and bones; the liver, fæces, and urine, may also contain traces until a late period. With regard to its presence in the *blood* in the human subject, slight traces of it were found in McMullen's case, four days after the withdrawal of the antimony, and in M. Marchal's case after eight days. On the whole, the blood appears to retain the metal in smaller quantity and for a shorter period than the other fluids and solids (ante, p. 49). In certain diseased states of the system, the complete elimination of the metal may require a period of from twenty-five to thirty days or longer; and antimony may therefore in some instances be found in the liver and urine at this date; but in a healthy subject, to whom only ordinary medicinal doses have been given, the antimony is quickly expelled. In accordance with Orfila's experiments, there is no reason to believe, that it would remain longer than fifteen or twenty days, after the last administration, in organs important to life.

In the case of John Parsons Cook, who died from strychnia administered by William Palmer, Dr. Rees and I found antimony in the blood, —in the coats of the stomach,—in the intestines, in the liver, the left kidney, and spleen. With the knowledge of the facts regarding absorption and elimination already related, we affirmed that this extensive diffusion and deposition of antimony in the body of the deceased was only reconcilable with the view that it was of *recent* and not of old introduction; that some portion at least had been taken within a few days, and probably the whole within a fortnight of death. The presence of the metal in the stomach and intestines, as well as in the blood, was inconsistent with the view that this antimony could have been in the body of the deceased for months or years. The whole quantity found was estimated at half a grain. We had clearly ascertained that antimony had not been prescribed by any medical practitioner in attendance on the deceased.

The following questions were put to me by Sergeant Shee in cross-examination at the trial of Palmer, in reference to the absorption and elimination of this metal:

The learned counsel, having attributed to Orfila experiments which Orfila had not performed, and results for which he was not responsible, attempted from these quotations to refute the conclusion which Dr. Rees and I had drawn in the case of Cook, namely, that some portion at least of the antimony had been administered to him recently before his death. It was suggested that MM. Millon and Laveran had found antimony in the bodies of animals between three and four months after the withdrawal of the antimonial compound (ante, p. 57, Ex. p. 4 and 5). Some antimony it was said had been found in the liver by these experimentalists, but the important facts that the antimony was chiefly accumulated in the bones and fat, and that there was none present in the stomach, intestines, and *blood*, were entirely suppressed from the knowledge of the Court by the learned counsel. In fact, with that boundless license which is too frequently assumed with impunity, in the defence of criminals, the counsel quoted so much as might appear to conflict with our evidence, and carefully withheld those material points which would have shown that the results of Millon and Laveran were wholly inapplicable to the case of Cook.

On an analysis of these experiments (ante, p. 56) it will be perceived: 1. That a large quantity of tartarized antimony (forty-five grains) was given to dogs in divided doses over a period of eight or ten days. Allowing that the rate of elimination is the same in a dog as in a human being, there was no evidence that the deceased (Cook) had received one-tenth part of this quantity of antimony, or that he had taken it regularly in divided doses over a period of ten days. 2. Dr. Rees and I had clearly detected antimony in the stomach and intestines, as well as in the *blood*. In the quoted experiments, these parts contained no antimony. 3. Under this long-continued use of antimony in dogs, Millon and Laveran discovered that the liver underwent a peculiar physical change, which certainly did not exist in the case of Cook.

No one who has perused the medical evidence given at this trial, can doubt that Cook was dosed with tartarized antimony before he was poisoned by strychnia. There was not a single fact to prove or render it probable that the antimony found in his stomach, intestines, and blood, had been there for many weeks or months; on the contrary, its presence in these parts was adverse to this theory; and the symptoms of vomiting stated to have occurred during his last illness, and shortly before his death, could only be adequately explained by that view which Dr. Rees and I had already adopted from the results of our analysis. In short our opinion, formed six months before the trial, was confirmed by the general and medical evidence given at the trial.

In allowing what a learned judge has designated "a considerable scope" to a defence, it is a serious question, whether under this head there should be included a *suppressio veri* and *suggestio falsi* in the misstatement of the results of the experiments, and in the misquotation of authorities. It seems reasonable, if a medical authority is allowed to be quoted at all on a question of science in order to support a certain view, that the whole of the results bearing upon the question at issue should be taken together. A medical witness under examination, may not be well acquainted with the work quoted, and by this dishonest mode of dealing with important scientific questions, his opinion, although well founded and in accordance with experience, may be easily made to appear inaccurate.

CHAPTER IV.

ABSORPTION, DEPOSITION, AND ELIMINATION OF OTHER METALS—SILVER, COPPER, LEAD, MERCURY—CADAVERIC IMBIBITION—DIFFUSION OF ARSENIC AND OTHER POISONS IN THE DEAD BODY—ERRONEOUS INFERENCES—ELIMINATION OF ARSENIC AND ANTIMONY BY THE STOMACH AND INTESTINES—ELIMINATION OF PHOSPHORIC ACID—ABSORPTION AND ELIMINATION OF PRUSSIC ACID—OF THE POISONOUS ALKALOIDS, MORPHIA, NICOTINA, AND STRYCHNIA—CONFLICTING RESULTS.

OTHER METALS.—In reference to the absorption and elimination of other metallic poisons there are but few facts on record. M. L. Orfila gave to animals salts of lead, mercury, silver, and copper in small doses for fifteen days or longer. The doses were so adjusted as not to produce very serious symptoms or death. The animals were killed at various periods and the viscera chemically examined. In all cases of recent administration the metals were found more or less deposited in the organs, provided a sufficient quantity had been given. Small quantities, as in the case of antimony, appear to be rapidly eliminated, and it is only after repeated doses that deposition is observed. *Silver* was not found to be eliminated by the urine. It was discovered in the liver five months after the cessation of the administration. It was not found after seven months, and in some instances not found after six weeks. The receptacle for the deposition of silver, after long periods of administration, is well known to be the skin, which is permanently discolored. The sulphate of *copper*, given under similar circumstances, was found in the urine only for the first three days after its administration had ceased, but copper was found in the liver, stomach (coats?), and intestines, for the long period of eight months afterwards. The skin and fat contained none, but M. Chevallier extricated it from the hair of a man who worked in a copper factory. In this case it was probably deposited mechanically in the hair. In an instance in which eighteen grains of sulphate of copper had been given to a child in one day and death took place in about twelve days, not a trace of copper could be found on an analysis of the bowels. (Casper's Leïchen Oeffnungen, 1853, p. 156.) MM. Danger and Flandin state that the salts of copper, when taken as poisons, may be more readily detected in the bronchial secretion than in the urine. (Ann. d'Hygiène, 1843, p. 452.) The heart and kidneys of animals poisoned by this metal contained no copper. Dr. Odling has informed me that he has found the liver to be a seat of deposition for copper, and that irrespective of poisoning a few ounces of liver will generally yield minute traces of this metal.

Lead was discovered in the urine for three days after the acetate had ceased to be taken, this substance having been given in small doses for a month. The liver, intestines, and brain yielded lead after eight months. The livers of two puppies suckled by a bitch gave traces of lead three or four days after the withdrawal of the acetate, and the liver as well as the stomach of the mother was found to contain traces of lead ten days afterwards. (Ed. Monthly Journal, March, 1852, p. 27, also Galtier's Toxicologie Générale, 1855, p. 18.) It is well known that in chronic poisoning by lead, as well as by mercury, the deposition of the metal shows itself in the gums at the point where they join the teeth, forming a dark blue line which remains for a considerable period. Dr. Wilson considers, from his experiments, that in chronic poisoning by lead the metal is more largely deposited in the spleen than in the liver and other organs. A mare drank daily for about six weeks water impregnated with carbonate of lead derived from some lead-works. The animal suffered from the usual effects of lead-poisoning, and died suddenly, about a fortnight after her removal from the spot, and after she had ceased to take the poisonous water. Lead was found deposited in the largest proportion in the different organs, according to the following order,—the spleen, liver, lungs, kidney, heart, and coats of the intestines. Hence, in suspected lead-poisoning he recommends that the spleen should be selected for analysis. (Ed. Monthly Jour. Med. Sci. vol. xiv, p. 389.)

It would appear that in chronic poisoning by lead, the skin and nails are also seats of deposit as well as the gums. In reference to some cases of lead-poisoning which occurred at Claremont, Dr. De Mussy states that, under the use of sulphur baths, the nails of the feet and hands of the persons affected were blackened, and similar spots came out on different parts of the skin. One of the patients came out from the second bath with the abdomen entirely black. (Dublin Quarterly Journal, May, 1849, p. 415.) It is probable that the urine and milk are channels of elimination for lead-poison. The metal has been frequently found in the urine of persons who were taking salts of lead medicinally; and in one instance I found it in milk drawn from a cow, while laboring under the effects of poison from licking lead-paint. (On the Absorption of Metallic Poisons, Guy's Hosp. Reports, 1841, No. xii.) In some remarkable cases of poisoning by lead among cows and sheep, which Mr. Brande and I were required to examine in Somersetshire, in 1855, the calves and lambs were extensively affected with the usual symptoms of lead-poisoning, probably imparted by the milk of the animals. There is great reason to believe that lead is a metal which, when once deposited, is only very slowly eliminated from the system. In a case of lead poisoning which occurred at Guy's Hospital in April, 1846, the metal was found in the liver. The woman died seven days after her admission. It was not improbable from the symptoms that the poison had been taken at least ten weeks previously. (Guy's Hospital Reports, 1846, vol. iv, p. 471.) In the year 1850, MM. Chatin and Bouvier, examined the liver and brain of a man who had been a worker in white lead, and who, owing to an attack of saturnine disease, had quitted the establishment two days previously. By the examination of the organs they procured clear evidence of the presence of the metal in larger proportion in the liver than in the brain. MM. Lassaigne and Chevallier made an examination in a similar case of the liver of the deceased weighing four and a half pounds,—of the brain weighing three pounds, and of the spinal marrow weighing about an ounce and a half. Traces of lead and copper were found in each of these organs, the copper being in larger proportion than the lead in the liver and brains, while the lead was in greater proportion in the spinal marrow. (Journal de Chimie Médicale, 1851, pp. 67, 134.) It is probable that the deposits of these metals in the tissues take place more readily where the salts have been taken in small doses.

Mercury has been repeatedly found deposited in the liver and other organs. In several cases I have detected it in the liver, in which it has been deposited as the result of small doses of calomel or blue pill taken shortly before death. In chronic poisoning it has been known to produce a blue line on the gums like lead, and it is probably retained by the tissues for a variable period. M. Orfila, in giving small doses of corrosive sublimate to animals, found mercury in the urine five or six days after its ingestion into the stomach; and in the saliva, when there was salivation, but not after the fifth day from the suspension of the poison. He obtained mercury from the liver, intestines, and kidneys; he did not find any in the fat, lungs, or bones, when the above-mentioned organs contained it. He considers that mercury is entirely eliminated in a month, and chiefly by the kidneys. This metal has been found to be eliminated by the milk. (Galtier, op. cit. p. 18.) In other experiments he found that mercury disappeared from the organs in about eight or ten days; in one instance it was found on the eighteenth day after the suspension of the mineral. In an experiment performed by Dr. Glover, half an ounce of corrosive sublimate was given to a dog and its œsophagus tied. The animal died in an hour. Dr. Glover examined the heart and eight ounces of blood, and found mercury therein. (Ed. Med. and Surg. Journal, Oct. 1842.) M. de Kramer states that he detected mercury in the blood of a person who had taken the sulphuret medicinally, but the particulars are not given. (Ann. d'Hygiène, April, 1843, p. 428.) According to Sobernheim, in cases in which mercurial preparations had been taken a long time medicinally for producing salivation, mercury was found in the blood, urine, and saliva. (Prakt. Toxikol. p. 243.) In a case of poisoning by arsenic, which occurred in March, 1858, I ascertained that the deceased had taken five grains of calomel in pills, twelve hours before death, and two grains of calomel in pills four hours before death. He died in twenty-six hours after he had taken the arsenic. Mercury was found abundantly in the contents of the stomach, the coats of the stomach, the contents and coats of the duodenum, in the bile, and in the substance of the spleen. Arsenic was found in small quantity associated with the mercury in the parts examined, excepting in the duodenum and the spleen. This last-mentioned fact is of importance, since some professed experts, who reason from ideas to facts, have gone so far as to assert that when two metals are in the body, if both are found in one organ, both must be found in all! In a case of poisoning from corrosive sublimate which occurred at Guy's Hospital in February, 1843, the patient was actively treated, but death took place on the fourth day. I examined the stomach and contents, five ounces of blood, the spleen, the serous liquids of the pericardium and the peritoneum, but there was no trace of mercury. (Guy's Hospital Reports, 1844, vol. ii, p. 25.) There appears to be, therefore, considerable uncertainty respecting the deposition and period of elimination of this metal. The time at which mercury, when once deposited in the soft organs, is entirely eliminated from the human body, has not been determined. According to an observation made by Dr. Gorup Besanez, it may remain in the liver for a year after the cause producing the deposition has been withdrawn. A woman who had worked for sixteen years in a mirror manufactory, was attacked with hydrargyrosis (mercurial disease), of which she died in a year. Although during this period she was entirely removed from the mercurial influence, the presence of mercury in the liver was nevertheless demonstrated with certainty after death. Mr. Burton, who quotes the case, reports another which was made the subject of analysis by himself. (Med. Times and Gazette, July 11, 1857, p. 84.) In this, the deceased woman had worked in a mirror factory until within a few weeks of her death. The whole of the liver was examined and mercury found in it.

These instances may be regarded as of an exceptional kind. The condition of body of persons who have breathed mercurial vapors for many years, can hardly be regarded as analogous to that of persons who have taken one or more doses of mercury as a poison or medicine. As there is no medicine so commonly taken as calomel or blue pill, it would be proper when mercury is found in the tissues, and before drawing an inference from its presence, to inquire whether any mercurial medicine had been taken shortly before death. We might otherwise be led into a grave error respecting the period during which this metal may remain in the system.

Diffusion of poisons in the dead body. Cadaveric Imbibition.—This subject has not hitherto received much attention from medical jurists; it is, nevertheless, one which may deservedly claim consideration when we are disposed to place reliance upon the presence of traces of poison in organs contiguous to the stomach. The effects of cadaveric imbibition have been greatly exaggerated. Observation shows that it is too limited in extent to affect materially the conclusions usually drawn from the detection of poisons in the tissues. When a dead body is examined for a mineral poison (such as arsenic or antimony) shortly after death, and it is found in the liver or other soft organs, it is a fair inference that it was deposited in them *during life*. When, however, the mineral is found in the viscera of a body long after death, as in a case of exhumation, then it may be objected that the poison has been imbibed by the tissues, and thus diffused through the body. This explanation of course presupposes that at the time of death the poison is somewhere in the body in a free state. It may be in the stomach and bowels, and from these parts it is supposed to spread as from a centre, by solution in the liquid portions of the tissues.

Orfila gives the following as the results of some experiments on this subject. On various occasions he introduced into the stomach or rectum either of dogs or of human beings after death, when the body was cold, from thirty to forty-five grains of arsenic *dissolved* in a pint of water. *Cadaveric imbibition* uniformly took place. The portions of liver, or other organs which touched the alimentary canal furnished arsenic, but those parts which were not in contact did not contain any poison. When the arsenic was thrown into the stomach and the body placed on its back, the poison was detected in the left half of the diaphragm, and in the lower lobe of the left lung, while none was procured from other portions of the diaphragm or right lung. (Toxicologie, i, 384, 1852.) Imbibition was here undoubtedly favored by the arsenic being dissolved in a large quantity of water. These facts are of importance, as showing that viscera suspected to contain poison, should not be placed together in a jar. The heart placed in contact with a stomach containing arsenic or antimony, may yield traces of this metal on analysis, although none were present when it was removed from the body. It is evident that this might lead to an erroneous opinion regarding the presence of the poisonous metal, as a result of absorption and deposition during life.

It has been suggested that arsenic might be introduced into the stomach of a person after death, that it might thence be spread to all parts of the body by imbibition or endosmosis, and in time equally impregnate all the tissues. Dr. Kidd has published some observations on this subject, which, although they can rarely have a practical bearing in well-observed medico-legal cases, are of some interest in medical jurisprudence. (See Observations on Imputed Poisoning, Dublin Quarterly Journal, 1850, vol. x, p. 78.) On introducing four and eight ounces of a solution of arsenic (ten grains to the ounce) into the dead bodies of a cat and rabbit respectively, he found in about a month that the poison had so diffused itself as to have impregnated adjacent organs. In the rabbit, the heart was thus impregnated with arsenic, the body of the animal having been suspended with its head downwards.

There can be no doubt that the solution of arsenic obeys the laws of endosmose and exosmose, observed in other liquids, and that it will permeate animal membranes, and affect liquids or adjacent solids containing liquids. If the mouth of a tube half filled with a solution of arsenic be covered tightly with a layer of animal membrane (bladder), and inverted in a jar of distilled water, so that the levels inside and outside are equal; it will be found that by endosmose there is a rapid intermixture of the two liquids. I have thus found the distilled water on the outside, impregnated with arsenic in an hour, not from any mechanical escape of the solution, but from actual passage through the membrane. In fact the level of the liquid inside the tube sometimes becomes higher, showing that water is actually added to its contents, and that there can have been no leakage. I have observed the same phenomena with solutions of chloride of zinc and sulphate of copper.

While the fact of transudation is undeniable, experiments of the nature of those performed by Dr. Kidd are so unlike anything that occurs in practice, that it is difficult to make a useful application of the results. In Dr. Kidd's experiments *eighty grains* of arsenic, *dissolved in eight ounces* of liquid, were placed in the stomach of an animal after death! In a human being at the time of death, there may not be one-tenth part of this quantity remaining in the stomach, and the arsenic in a pulverulent, not in a dissolved form. After various periods of interment, up to two years, I have seen the arsenic in yellow masses fixed in the coats of the stomach and bowels, dyeing them of a deep yellow color. There is every obstacle to diffusion in these cases by rapid conversion to sulphuret, and the fixation of the arsenic in an insoluble form. Again, the distribution of the arsenic is commonly wholly unlike that which would follow from the mechanical causes here supposed. More may be found in the rectum than in the intermediate tract of intestines; none will be found in the spleen, while there may be a larger quantity in the heart, and none may exist in the diaphragm, although the body may have been placed in a horizontal position in the grave. As to the liver, the inside will contain as much as the outside, and the right side as much or more than the left side, which is adjacent to the stomach. I have found much arsenic in the bile and scarcely a trace in the coats of the gall-bladder, and at the same time none in the stomach. In a recent case, where the body had been buried two years, arsenic was found in the intestines, but the mesentery on which they were lying did not contain a trace. The theory of this source of the arsenic in the tissues assumes that there are no symptoms in the living, or appearances in the dead body, to aid the judgment,—that the person dies from natural causes, and that soon after death some person injects into the stomach, for some purpose, a large dose of arsenic in solution, and contrives to suppress all evidence regarding the exercise of this malicious ingenuity. Such a condition of things is, I believe, impossible, without certain detection in the majority of cases calling for medico-legal examination; and a proper attention to the symptoms, appearances, extent of diffusion, and proportionate distribution of the arsenic, in any one instance, would enable an experienced witness to determine whether there had been administration as well as absorption during life, or injection and imbibition after death. I have had occasion frequently to consider this question in cases that have come before me, and I may observe, that in every instance, the facts have been wholly opposed to, and inconsistent with such a theory. The same observation applies to the diffusion of antimony and other poisons. (See for some remarks on this subject, a paper by Dr. Geoghegan, on Arsenical Poisoning in Dublin Quarterly Journal.) Orfila has satisfactorily proved that the supposed difficulty arising from the imbibition and diffusion of poisonous substances, placed in the body after death, is rather of a theoretical than of a practical kind. (Toxicologie, Ed. 5ème, i, 55.)

Arsenic and antimony, when absorbed and deposited in the organs during life, are so deposited as to be in part at least soluble in water. In two instances in the human subject I have thus separated arsenic from the liver by boiling a portion of it in water and testing the filtered liquid. The liver of an animal poisoned by arsenic and recently killed, also yielded this poison to water. Absorbed antimony appears to be more readily separable from the tissues (liver, spleen, and kidneys) by boiling water, than arsenic. This I have verified on the dead body of a person poisoned by antimony. The fact that some portion of the metallic poison was deposited in a soluble form was noticed by Orfila (Toxicologie). It affords no support to the hypothesis of diffusion after death, as it occurs in recent cases of poisoning in which there has been no time for diffusion.

The presence of poison (such as arsenic or antimony) in the stomach or bowels has generally been taken to indicate a recent administration by the mouth or rectum. If the poison be in some quantity, and in a solid or liquid form in the contents, this inference is justifiable, but if only in traces in the coats or in the mucus of the stomach, it may be the residue of a quantity taken by the mouth some days previously. It has been said that the mucous membrane of the stomach is itself an eliminating surface. Experiments on animals appear to show that some poisons introduced by wounds or into the blood may be found in the coats and mucous fluids of the stomach. I have elsewhere referred to experiments of this kind (ante, p. 58). It must not be supposed, however, that this mode of elimination by the stomach or bowels, assuming that it takes place in the human body, is such that it will account for or explain the presence of a large quantity of poison in the contents of these viscera, or in any case for the presence of arsenic, antimony, or other poisons, in a solid or tangible form, or for the presence of a larger quantity of a soluble poison in these parts than is found in all the other viscera taken together. Any of these conditions would indicate a recent introduction of a poison by the mouth or by the rectum. If the poison be contained in the stomach and intestines and not in the other viscera, and at the same time there has been no application to a wound or ulcer, it is reasonable to presume that its presence is the result of ingestion into these parts, and not of elimination from the mucous surface. The remarks elsewhere made with respect to antimony (p. 59) apply to arsenic and other poisons.

But little is known concerning the elimination of the metalloidal poisons and their salts. Some remarks have been elsewhere made on the absorption and elimination of iodide of potassium. Dr. Böcker found that *phosphoric acid* was rapidly eliminated from the stomach without passing into the intestines. He gave to a dog fifty-four drops of phosphoric acid (containing eight grains of anhydrous acid), diluted with three parts of water. In half an hour afterwards barely a trace could be found in the stomach, and there was none in the intestines. He considers that in a diluted state phosphoric acid rapidly enters the blood, and in combination with alkalies, it is speedily eliminated in the urine. From experiments on himself, he concludes that in a healthy state of body, as much as one hundred drops, or fifteen grains of anhydrous phosphoric acid are entirely eliminated, or removed in the urine, in from twenty-four to forty-eight hours after the acid has been swallowed. (Reil's Journal für Toxikologie, 1856, H. i, p. 87.) This may account for the occasional absence of any trace of phosphorus in the bodies of persons poisoned by that substance.

Prussic Acid.—Poisons, which are of a liquid and volatile nature, enter the blood, are diffused through the body, and eliminated with great rapidity. Prussic acid and alcohol furnish instances of this rapid elimination. Müller states that a liquid poison brought into contact with a wounded surface, may be distributed through the system by absorption in from half a minute to two minutes; but Mr. Blake has inferred from his experiments that a poison may be diffused through the body in so short a period of time as *nine seconds;* and he states that an interval of always more than nine seconds elapsed between the introduction of a poison into the capillaries or veins, and the appearance of its first effects.

Kramer discovered prussic acid in the blood of an animal which died in *thirty-six seconds* after its administration in the ordinary way. Mr. Waterworth, formerly a pupil at Guy's Hospital, gave to an animal a fatal dose of prussic acid, and in less than a *minute* afterwards, when all signs of life had ceased, he made an opening into the chest, and tested the warm vapor which escaped. In this vapor he clearly detected prussic acid. This appears to establish the correctness of Müller's view in respect to the very rapid diffusion of this poison. These volatile poisons, like the soluble gases, appear to be chiefly eliminated by the lungs. So long as life remains, the peculiar odor of the poison is plainly perceptible in the breath. That portion of the poisonous compound which undergoes no change in the blood, is rapidly carried off. Dr. Lonsdale found in his experiments on animals, that when life was prolonged beyond *fifteen minutes*, the odor of prussic acid could not be perceived either in the blood or in the cavities; but if the death of the animal took place within a few minutes after the administration of the poison, then the odor might be detected in the cavities of the body for eight or nine days afterwards. (Ed. Med. and Surg. Jour. vol. 51, p. 52.) The power of detecting this volatile poison by its odor or vapor in a dead body, depends, therefore, on the length of time a person survives the effects. If the dose be small, and death a slow consequence, then the greater part, if not the whole, may be eliminated. In general, however, the dose is large, and death takes place speedily; hence, the poison may be found, unless owing to its great volatility it has entirely passed away before the inspection is made.

After death, if the body be examined recently and it has not been too long exposed, prussic acid in the absorbed state may be readily detected. I have thus found it in the blood, coats of the stomach, liver, and other parts. Dr. Lonsdale relied on the odor for determining its presence or absence, but this is subject to many fallacies; a method of fixing the acid by chemical reagents is now exclusively resorted to. This poison, unlike arsenic and antimony, impregnates all parts of the body at once, and being volatile, it does not remain fixed in the organs like these metallic substances. If found long after death, the discovery of it must depend on a large quantity remaining in the body at the time of death. As to the loss of this poison, I may remark that I have found it to escape when covered with a stratum of oil, and the vessel was tightly closed with bladder. Hence, in preserving viscera for analysis, a sheet of tin-foil should be used as a covering to the bladder.

The Alkaloids.—There can be no doubt that these powerful agents, of which morphia and strychnia may be taken as types, are absorbed into the blood, and diffused through the system like other poisons. The statements regarding their deposition in the viscera, and their elimination are, however, of a conflicting kind. In a remarkable case of poisoning by nicotina, in 1847, M. Stas had announced the discovery of this alkaloid in the tissues; but it is questionable whether this was not some portion of the nicotina which had been imbibed by the organs, rather than that which had been absorbed and deposited in them. "On three occasions," M. Stas observes, "during a period of six years, I have discovered the alkaloids in cases of poisoning: in 1845, at Bruges, morphia was detected in the viscera of a body after an interment of thirteen months; in 1847, morphia was also detected in the viscera of another body; and about this period, I detected aconitina in a suspected liquid which had become considerably changed (profondément altéré)." The quantity of morphia taken by the deceased persons, the period which they survived, the quantity found in the bodies, the viscera in which it was detected, and the tests upon which M. Stas relied to swear positively to the presence of morphia and aconitina, are not mentioned. Although no cases are given, or facts stated, M. Stas sums up his memoir by the general remark—"I have applied the principles just laid down (by his method of research) to morphia, codeia, strychnia, brucia, veratria, emetina, colchicina, aconitina, atropia, and hyoscyamia; and I have been able, without the slightest difficulty, to separate these different alkaloids when previously mixed with foreign matters." These results, however, cannot be taken as referring the separation of the poisons above mentioned (deposited as a result of absorption) from the viscera of human beings or animals which had taken them during life; for on this subject there is no account of a single experiment. The analysis refers to the separation "of strychnia and brucia from nux vomica, veratria from the extract of veratrum, emetina from the extract of ipecacuanha, colchicina from the wine of colchicum, aconitina from an aqueous extract of monkshood, hyoscyamia from a very old extract of henbane, and finally atropia from an old tincture of belladonna." (Flandin, Traité des Poisons, iii, pp. 144 and 255, 1853.) Facts of this description have a pharmaceutical interest, but until the results have been verified by repeated trials in the dead body, they are of very little value to a medical jurist. Some of the poisons which are here mentioned will destroy life in a minute fractional proportion of a grain; and no process, however delicate, can make up for a small quantity of poison distributed by the circulation through an enormous mass of animal matter. For example, Mr. Morson informs me that the one-hundredth part of a grain of aconitina prepared by him, is a full medicinal dose, and that the fiftieth part of a grain might prove fatal to a human being. When diffused through twenty-eight pounds of blood, and spread through the whole of the tissues of the body, it would be about as easy to obtain aconitina in a dry and tangible form from the liver, as to procure from this organ in a fatal case of hydrophobia or of serpent-bite, a visible quantity of the poison of rabies, or of the ophidian poison—echidnine.

Morphia.—Among the numerous fatal cases of poisoning by *opium* in this country, no chemist of repute, so far as I can ascertain, has announced the detection and separation of *absorbed morphia* from the tissues. M. Bouchardat states, that he has found morphia as well as atropia and daturia to be speedily eliminated in the urine when these alkaloids have been prescribed in medicinal doses. He employed for this purpose a peculiar test composed by weights of one part of iodine, two parts of iodide of potassium, and fifty parts of water. It gives a precipitate in the urine when an alkaloid is present, but it does not serve to distinguish one alkaloid from another. The urine of rabbits fed on the leaves of belladonna, gave a precipitate of atropia on the addition of the test. The urine of opium-eaters gave with the test a precipitate of hydriodate of morphia; but in this case, unless a large excess of the test was used, the precipitate at first formed was redissolved. In a case of poisoning by opium which was rendered doubtful by the symptoms, the use of the test revealed the presence of morphia in the act of elimination by the urine. This reagent indicates that in poisoning by opium, morphia is very rapidly eliminated. A woman attempted to poison herself with two drachms and a half of laudanum. In three hours after she had taken the poison, the urine gave with the above reagent an abundant precipitate of morphia. To test the rapidity of elimination, one grain and a half of crude Smyrna opium, mixed with food, were given to a dog. In three hours there was a slight precipitate of morphia on adding the test; in five hours there was a well-marked precipitate; in seven hours it was still perceptible, but in eight hours and a half from the time at which the dose was given there was not a trace. A repetition of the experiment on the same dog produced like results. When an interval of twenty-four hours was allowed to elapse, there was no precipitate. Hence, the alkaloid may not be found unless sought for within from seven to ten hours after the poison has been taken. To insure precipitation, the supernatant urine should be poured off and a fresh portion of the test added. The urine of a patient taking three-quarters of a grain of opium in two doses daily, yielded faint traces of morphia; but it was only clearly precipitated when the test was added in great excess to the urine recently voided. When carbonate of ammonia had been produced in the urine as a result of decomposition, the test failed to act. A female took one grain and a half of crude opium at night. A small quantity of urine voided at night gave a slight precipitate of morphia, but that which was passed at noon on the following day, gave no precipitate. (Supplement à l'Annuaire de Thérapeutique, 1856, p. 223.) These results confirm the observations of Bernard, namely that the poisonous alkaloids are rapidly carried out of the body. In about twelve hours after a small medicinal dose has been taken, none may be found.

M. Bussy found in giving to a dog an aqueous solution of extract of belladonna, that in fifteen minutes there was a perceptible dilatation of the pupils of the eyes, a clear proof that atropia had been absorbed and had so far saturated the blood as to paralyze the ciliary nerves. (Ann. d'Hygiène, 1847, ii, p. 418.)

Strychnia.—The same remark applies to *nux vomica* as to opium. Notwithstanding the large number of fatal cases of poisoning by this substance, which have occurred in England, and the fact that death is uniformly caused by the strychnia which is contained in it, no chemist has yet succeeded in separating the alkaloid strychnia in an absorbed state from the blood, tissues, or soft organs of the body. How is this to be explained? Men who have shamelessly stated on oath that there is no poison so easy to detect, have, when cases have actually occurred to them, shrunk from the responsibility of testing their theories by facts, and have declined to place in jeopardy their reputations for accuracy by endeavoring to extract the alkaloid from the liver of a human being poisoned by nux vomica. Knowing well that an animal that dies from nux vomica dies from absorbed strychnia, they have contented themselves with seeking for nux vomica powder and extracting strychnia from it. Even with regard to the alkaloid, strychnia, itself, among fourteen fatal cases which had occurred up to the time of the trial of William Palmer, in May, 1856, chemical science was a blank. In no one instance had strychnia been obtained from the tissues of a person, poisoned by it, and in the greater number of instances it had not even been found in the stomach in an unabsorbed state, *i. e.*, as the residue or surplus of the poisonous dose, which had caused death.

In the year 1827, Vernière showed by an ingenious physiological experiment that the poison of nux vomica (strychnia) entered the venous blood by absorption, and that the blood thus impregnated, when transfused into another animal, produced the usual symptoms of poisoning. It is probable that if a very large dose of nux vomica could be given to one animal, and, while laboring under its effects, a sufficient quantity of blood could be taken from it and transfused into the body of another, it might be found that this liquid would act as a poison and cause death. There are, however, insuperable obstacles to the performance of such an experiment; because if a large dose of poison be given to the first animal, it may die before a sufficient quantity of blood be transfused from it. If a small quantity of poison be given, or a small quantity of blood be transfused, no fair inference could be drawn from the results; if a large quantity of blood be transfused, this might cause the death of the animal which lost the blood, and yet not be sufficient to produce fatal effects in the other.

The rapidity with which strychnia is absorbed and diffused through the body, varies probably according to many circumstances. On the fact of its diffusion, there is one set of experiments by Mr. Blake; he found, on introducing the nitrate of strychnia into a vein, that the action of the poison on the spinal cord was manifested by tetanic convulsions in sixteen seconds in the horse, in twelve seconds in the dog, in six and a half seconds in the fowl, and in four and a half seconds in the rabbit. Severe symptoms cannot be produced until the poison is diffused through the circulation; and the more rapidly it enters the blood, the more speedily do the effects appear. This shows how largely absorption must be concerned in the operation of the poison. Dr. Christison killed a dog in *two minutes*, with the sixth part of a grain dissolved in alcohol, injected into the chest; and a wild boar was killed in *ten minutes* with one-third of a grain. An instance has been privately communicated to me, in which a man died in *ten minutes* from a dose of ten grains! This is the most rapid case of death yet known; and there must have been here very speedy absorption and diffusion. Dr. Harley injected one-twelfth of a grain of acetate of strychnia in solution, into the jugular vein of a full-grown dog; in *four seconds* the animal became tetanic, and in twenty-eight minutes it died.

In these cases the absorption of the poison is inferred from the physiological effects produced, and not from the chemical demonstration of its presence in the blood and tissues.

With respect to the detection and separation of the poison from the blood and tissues, the results are not satisfactory. We look in vain in the treatises of Orfila, Christison, and the more recent works of Galtier and Flandin, Casper, Otto, and Böcker, for any instance in which *absorbed* strychnia has been detected either in the human being or in animals; and this remark may be extended to the other alkaloids generally. Since recent events have directed particular attention to this subject, it has been announced that in numerous experiments strychnia has been detected in the blood and tissues.

Dr. Ogston states, that, in a case of poisoning by strychnia, he found traces of the poison in four ounces of blood. Dr. McAdam states that he detected strychnia in the tissues of a cat which died in fifty-six minutes after a quarter of a grain had been given. This gentleman also states that he found it in the urine voided by a dog, only nine minutes after half a grain had been given to the animal. The dog was not at the time suffering from symptoms of strychnia-poisoning! This result shows that the urine is one medium of elimination, and that the removal of the poison from the system by this channel may, in some instances, go on with extraordinary rapidity. (Guy's Hosp. Reports, Oct. 1856, p. 393.) In a horse killed in two hours by thirty-two grains given in divided doses, Dr. McAdam found strychnia in the muscles, blood, and urine contained in the bladder. He did not detect it in the liver, lungs, spleen, kidneys, or heart. (Pharmaceutical Journal, August, 1856, p. 126.) Dr. Cowan, of Glasgow, poisoned three dogs, by giving to each of them one-quarter of a grain of strychnia. Dr. Anderson found traces of the poison in the liver of one dog, and Dr. Easton found it in the urine of another. In the Guy's Hospital Reports for October, 1856 (p. 270), I have fully detailed the experiments on this subject which formed the basis of the evidence for the prosecution and defence in the case of Reg. v. Palmer (Central Criminal Court, May, 1856). One of the witnesses retained for the defence on that occasion, professes to have since found strychnia deposited in the *bones* of animals as a result of absorption. (See Pharmaceutical Journal, April, 1857, p. 497.)

On the other hand, Dr. Harley, of University College, examined the blood taken from the heart and large vessels of a dog killed by the twelfth part of a grain of acetate of strychnia injected into the jugular vein. The animal was tetanic in four seconds, and died in twenty-eight minutes. The blood, on chemical analysis, yielded no strychnia. Mr. Horsley, of Cheltenham, examined the blood and tissues of the dog which he poisoned (ante, p. 42), but no strychnia could be detected. He sent me a portion of the blood of the dog, equivalent to about two ounces, and, on analysis, I did not find in it the slightest indication of the presence of strychnia. Dr. De Vry, of Rotterdam, poisoned a dog with a solution of nitrate of strychnia introduced into a wound, and immediately after death he examined four ounces of the blood of the animal, but not the least trace of strychnia could be found in it. In another experiment in which a dog was poisoned in four days by half a grain of strychnia, in divided doses, the chemical analysis led to a perfectly negative conclusion, not only in the blood and tissues but in all parts of the body. (Pharmaceutical Journal, March, 1857, p. 450.) Dr. Crawcour, of New Orleans, gave half a grain of strychnia to a rabbit: the animal died in half an hour. No trace of strychnia could be found in any part of the body. (New Orleans Medical Gazette, Sept. 1856, p. 387.) Dr. Penny, of Glasgow, examined the brain and spinal marrow of a dog, poisoned by strychnia, without detecting a trace of the poison. It has been asserted that strychnia is eliminated by the urine; and experiments on animals, in reference to this point have been cited. In a case of poisoning by strychnia, which occurred to Dr. Geoghegan, of Dublin, in 1856, thirty ounces of urine passed by the patient from the fifth to the thirty-first hour after symptoms had commenced, when carefully analyzed, did not yield any trace of strychnia. Dr. De Vry examined the urine passed in twenty-four hours by a patient taking half a grain of nitrate of strychnia daily, medicinally, but he did not find in it the least trace of the alkaloid. (Pharm. Jour. March, 1857, p. 450.) Observations made on the human subject do not support the view that absorbed strychnia is either constantly eliminated by the urine or always deposited in the tissues so as to admit of separation by chemical processes after death. A case of some interest occurred to Mr. Wilkins, of Newport, in the Isle of Wight, in February, 1857. A gentleman died, under the usual symptoms, in about six hours after taking three grains of strychnia for the purpose of self-destruction. The long period which he survived was most favorable for the diffusion and deposition of the poison. The blood and the heart were examined by the late Mr. Scanlan and myself; portions of the liver and lungs were examined by Dr. Christison and Dr. Douglas Maclagan, of Edinburgh; and one kidney was examined by Dr. Geoghegan, of Dublin. The result was, no trace of absorbed strychnia was detected in any one part.

It is quite clear, therefore, from these negative results, obtained by gentlemen many of whom could have had no intention to uphold a foregone conclusion, that strychnia is one of the alkaloids which in some cases is either speedily eliminated, or, if deposited in the tissues, so altered in its nature, or diffused in so small a quantity, that the most refined chemical processes at present known cannot separate it. To assert that the minutest quantity of this poison can always and under all circumstances, be detected in the human body, because an almost infinitesimal quantity can be detected *out* of it, is not merely a simple absurdity but an untruthful statement, calculated to mislead a jury and to deceive the public. Certain analysts may never fail to detect absorbed strychnia in cases in which they *know* it has been administered; their sagacity is of the *ex post facto* kind; but, looking to what has been discovered respecting the absorption, deposition, and elimination of such poisons as arsenic and antimony (so easy of detection), it is only reasonable to suppose that strychnia is not an exception to the variations to which they are known to be subject,—namely, that it may be found in one organ and not in another, and that at one time the body may yield evidence of its presence, while at another time there may be no such evidence forthcoming. One of the most recent experimentalists on this subject, states that the alkaloids, strychnia, nicotina, morphia, and prussic acid, are rapidly eliminated. (Leçons sur les Effets des Substances Toxiques, par M. Bernard, 1857, p. 100.) According to his view, these alkaloids are either not deposited in the organs, or are speedily removed from them,—a conclusion which is supported by the negative results of the analysis made by Dr. Christison, Dr. Geoghegan and myself, in the case referred to me by Mr. Wilkins, as well as by the experiments of Dr. De Vry, Mr. Horsley, and others.

Before the year 1839, arsenic and antimony had not been found in the blood and tissues of persons who had been poisoned by these substances, and it is not improbable that improvements in chemical research may enable a chemist hereafter to separate strychnia and other alkaloids from the blood, with the same facility as arsenic or antimony, provided these organic alkaloids are retained in the system and are as little liable to change as the two metals above mentioned. (See post, *Causes of Non-detection of Poisons.*)

In reference to the detection of the other alkaloids in an absorbed state, there is an absence of facts. That they enter the blood by absorption is placed beyond doubt; but whether, when there, they are partially changed, or deposited unchanged in the organs, has not yet been satisfactorily established by experiment. I have elsewhere published some observations on this subject (Guy's Hospital Reports, October, 1856), and the researches of Dr. De Vry, of Rotterdam, have more recently led him to the conclusion that that part of the alkaloid strychnia which acts mortally, is decomposed in the living body. (Pharm. Journal, March, 1857, 451.) The examination of a large number of cases in the human subject can alone lead to a satisfactory settlement of this important question. A court of law has to deal with the fact and not with the theory. Absorbed strychnia is stated to have been found in the tissues of animals on various occasions, while other chemists have not succeeded in finding it in human beings or in animals to which it had been administered, and where it was clearly the cause of death. It is obvious, from statements which have been published, that those who assert they have invariably found absorbed strychnia, are quite prepared to swear to its presence in the dead body from results and in quantities which admit of no kind of corroboration whatever. When this sort of evidence is adduced for the purpose of leading a man to the scaffold instead of saving him fróm it, we shall be able to judge whether it will equally receive the sanction of a court of law, and the temporary sympathy of the public. At the same time, without denying to these ardent experimentalists the privilege of swearing to any extent they please, so far as their own experience is concerned, it is unreasonable to expect that their oaths should be allowed to neutralize or bind the experience of others.

CHAPTER V.

ABSORPTION, DEPOSITION, AND ELIMINATION OF ALCOHOL—LOCAL EFFECTS OF POISONS—CORROSIVE SUBSTANCES—VEGETABLE POISONS—REMOTE OR SYSTEMIC ACTION OF POISONS—THE CAUSE OF DEATH—CHANGES PRODUCED BY VARIOUS POISONS IN THE BLOOD—ACTION OF PRUSSIC ACID AND OXIDE OF CARBON—PRODUCTION OF POISONS IN THE BLOOD—EFFECTS OF DEPOSITION IN THE ORGANS—ELIMINATION OF SULPHURETTED HYDROGEN BY THE LUNGS.

Alcohol.—THE absorption, deposition, and elimination of *alcohol* was established many years since by the experiments of Dr. John Percy. Dr. Cooke and Dr. Ogston, of Aberdeen, had announced that they had found an alcoholic liquid effused in the ventricles of the brain of drunkards. Dr. Percy's observations do not confirm this statement, but they show that alcohol is conveyed by absorption and temporarily fixed in the brain and liver. Six ounces of alcohol (S. G. ·850) injected into the stomach of a dog, killed the animal in an hour and ten minutes. The brain yielded by distillation with water a small quantity of liquid, having the properties of alcohol. The blood contained none. In a second experiment two ounces of alcohol were injected, and in an hour and twenty minutes the animal was destroyed. The brain, blood, urine, and bile yielded an appreciable quantity of alcohol. In other experiments of a similar kind, alcohol was found generally in the brain, but not uniformly in the blood or urine. In a dog which lived twelve hours after the injection of two ounces, alcohol was not found in the brain or blood, but it was found in the bile and contents of the stomach. In the case of an adult who died from drinking a quantity of rum, alcohol was separated by Drs. Christison and Percy as a result of distillation from the substance of the brain; but in a case in which a man survived three days, none was found. The rapidity with which alcohol is absorbed and diffused is indicated by the following experiment. About two and a half ounces of alcohol (·850) were injected into the stomach of a dog. The death of the animal was almost instantaneous; both circulation and respiration were simultaneously arrested. The brain was distilled in sixteen minutes after death, and a small quantity of alcohol obtained; the blood also yielded alcohol, and an alcoholic smell was perceptible in the viscera of the chest and in the brain. It is remarkable that although the animal died in less than two minutes, nothing was found in the stomach. This experiment shows that alcohol penetrates very quickly to all parts of the body, and it meets a difficulty which has been raised respecting the action of poisons by absorption. But for the discovery of alcohol in the blood and brain, this is precisely a case in which death would have been set down to sympathy. (Experimental Inquiry on Alcohol in the Brain, by John Percy, M.D. 1839, p. 61.) Elimination appears to be very rapid. It takes place chiefly by the lungs,—a fact indicated by the odor of the breath in man and animals. The following experiment on a dog shows how soon all traces of alcohol may disappear. Three ounces, of a specific gravity of ·850, were injected into the stomach. The animal died in about eight hours; not a trace of alcohol was detected in the brain, blood, bile, and contents of the stomach. (Op. cit. p. 67.) Thus, then, this was a case of death from poison after complete decomposition and elimination; in eight hours the whole of the poison had disappeared (ante, p. 48). As a fact indicative of the uncertainty of the chemical results in alcoholic poisoning, another experiment may be mentioned. Eight ounces of alcohol (at ·850) were injected in two doses into the stomach of a dog. The animal was killed by prussic acid in two hours and a half. No alcohol was found in the brain. These experiments show that the absorption and elimination of alcohol take place rapidly, and that in a few hours all traces may disappear, and that in spite of this disappearance, an animal may die from the effects. Dr. Percy did not find alcohol in the fluid sometimes effused in the ventricles; but he believes that it is generally diffused through the substance of the brain. It may be found occasionally in the blood, but it may be separated with great facility from the bile and the liver. It does not appear to be eliminated to any extent by the urine; he separated it only once from the urine of dogs, and he once obtained evidence of its presence in the distillation of five ounces of human urine. The test employed for the detection of alcohol in these experiments was distillation with or without water, rectification by carbonate of potash, and the combustibility as well as a power of dissolving camphor in the liquid obtained. According to the researches of MM. Bouchardat and Sandras, alcohol passes undecomposed into the blood, but it is not eliminated by any secretory organ; a small portion only escapes by the lungs, and may be collected in the gases and vapors exhaled. If the quantity absorbed be large, the arterial blood retains the color of venous, and may give rise to asphyxia. The oxygen received into the lungs during respiration, transforms a portion of the alcohol into water and carbonic acid; but acetic acid may even be a product of intermediate conversion. Alcohol and the compounds derived from it disappear very rapidly from the system. (Comptes Rendus, 1846.) The tendency of alcohol appears to be rather to diminish the secretion of urine. (Med. Gaz. vol. xxxviii, p. 430.)

Local effects of poisons.—This may be regarded as a summary of the facts at present known regarding the absorption, deposition, and elimination of poisons. There are some substances of the absorption of which there can be no doubt, although their presence in the blood and tissues may not be demonstrable either physiologically or chemically; but, considering the facts already known regarding this process, it is reasonable to infer that all poisons sooner or later enter into and circulate with the blood. Is this absorption, however, in all cases, a necessary antecedent of their fatal action? In some instances, as, with reference to corrosive poisons, the mineral acids, the nitrate of silver, and other substances which produce local chemical changes, although the substance is sooner or later absorbed, absorption does not appear to be necessary to the poisonous action. The fatal effects of these substances can only be ascribed to the extensive local changes produced in the form of inflammation, ulceration, gangrene, or chemical corrosion. Some poisons have a strong local action as irritants or corrosives, and at the same time are taken up by the absorbents, and probably produce their fatal effects by absorption, *e.g.* phosphorus and cantharides. Arsenic and antimony, although capable of producing by local action severe inflammation of the stomach and bowels, must be regarded as poisons acting by absorption. The blood and the tissues are everywhere penetrated by them before death; a few grains of either substance placed in a bag and introduced into a wound in the cellular membrane, or placed on an ulcerated surface, will produce all the effects of poisoning without local change, and persons have died from the effects of arsenic and antimony taken by the stomach, in cases in which few or no morbid changes were observed in this organ or in the intestines. On all occasions, therefore, arsenic may be regarded as producing its noxious effects by poisoning the blood. Inflammation of the stomach or bowels may or may not accompany this action; but until the arterial blood is poisoned as a result of absorption, there is no reason to believe that it operates fatally. The symptoms are much the same in character, whether the poison be absorbed through a wound or through the mucous membrane of the stomach. The only difference observed will be in the rapidity of their occurrence and progress.

Poisons susceptible of absorption, which have no local chemical action, may produce local effects on the nerves of a part. This effect may be manifested through the comparatively non-absorbent skin. It has been stated, but on doubtful authority, that a person has died from the effects of anhydrous prussic acid dropped on the unbroken skin. If so, death may have been really caused by the respiration of the vapor, and not by absorption or local action. I have elsewhere made some remarks on this subject (see ante, p. 35). The local effect here alluded to may be, and generally is, confined to the sentient extremities of the nerves only, manifested by the occurrence of tingling numbness or paralysis. It is well known that aconitina, morphia, and prussic acid are capable of affecting the nerves, if they remain sufficiently long in contact with a part; and many experiments have proved that even the nerves supplying the hollow viscera, through which sensation is not manifested, are equally susceptible of this local action. Opium applied directly to the intestines has been known to put an end to their peristaltic motion; and the same phenomenon was accidentally observed by Addison and Morgan in their experiments with the ticunas poison. There are, however, no conclusive experiments to show that the impression indicated by paralysis of motion or sensation, is actually conveyed through the nerves. On the contrary, the impression appears to be essentially local, or restricted to the part touched by the poison. The numbness produced by prussic acid on the skin is confined to that part only to which the acid is directly applied: it does not appear to extend in any perceptible degree beyond this part. The experiment of M. Serres with liquid ether have furnished similar results. Having laid bare the nerves of the thighs of several rabbits, and placed them in contact with a sponge dipped in ether, it was observed that the sensibility of the nerve was destroyed at the points immediately in contact with the ether, but the entire sense of feeling remained in the portion of nerve above the point immersed in ether. In order to determine how far exposure to air affected the results, two nerves were laid bare; one was immersed in ether, and the other simply exposed to the air: in five minutes the nerve in ether was dead to all sensation, even on the application of pincers, while the nerve exposed to air retained all its sensibility and power of exciting contraction. The application of tincture of nux vomica, strychnia, or its salts, to the nerve in ether did not produce the least sign of sensibility or power of contraction. (Academy of Sciences, February 8, 1847.) Experiments of a similar kind, and with like results, have been performed with chloroform. Hence this liquid has been much used as a local anæsthetic. (Med. Gaz. vol. xliii, p. 79.) The local action of belladonna in paralyzing the ciliary nerves and causing dilatation of the pupil is a well-known physiological fact. It produces dilatation of the pupil of one eye without disturbing vision or causing dilatation of the pupil of the other eye. Müller considers that this is a local action on the nerves: if so, it differs from other local actions in being transmissible to a certain distance by the nerves. Some physiologists believe that absorption is necessary to this action, and when we consider that dilatation of the pupils is a well-marked symptom of poisoning by belladonna, if taken into the stomach, this belief appears to be well founded. In the general action of the poison, however, both pupils are dilated, while in the local application of it, only that pupil is dilated corresponding to the eye to which the belladonna is applied. The difference between the local action indicated by physical change, and that which is unaccompanied by any such change, is, that in general the former, being chemical, takes place equally in the dead and the living—the latter, which may be described as physiological, is observed in the living subject only.

The application of strychnia to a nerve does not produce the effects of poisoning, and a nerve cannot serve as a channel for the introduction of the poison into the system. Dr. Harley, in one experiment, cut through all the tissues of the hind leg of a frog, excepting the nerve, which still served to attach the limb to the body. The leg thus hanging only by the nerve was introduced into a solution of strychnia; but not the slightest symptom of poisoning ensued. In another frog the tissues of a hind leg, including the nerves, were cut completely through, with the exception of the femoral artery and vein, which alone kept up a communication between the body and amputated limb. Symptoms of poisoning appeared soon after the limb was placed in the same solution of strychnia. (Physiological Action of Strychnia, 1856, p. 6.)

The last-mentioned experiments show that strychnia, although it most powerfully affects the nervous system, exerts no local action on the nerves. They further prove that the channel by which the body is poisoned and the symptoms are manifested is *the blood.* The poison of the viper, and of poisonous serpents generally, appears to have a remarkable local action. When a drop of this poison, which is a clear viscid fluid, is introduced into a wound, the edges of the wound become black, and the part inoculated, as well as the whole limb, begins to swell.

There is at the same time a discharge of a bloody liquid. (Bernard, op. cit. p. 391.)

It was formerly supposed that the noxious influence exerted by poisons on organs at a distance from the part to which they were applied was the result of a local effect transmitted through the nerves,—that the poison acted directly and exclusively on the nervous system. This was termed an action by sympathy. It was believed that some of these agents produced their effects with a rapidity too great to render it possible for them to be absorbed and distributed through the blood. Animals have been killed in two or three seconds by prussic acid; and conia, as well as some other poisons, have been found to act with equal rapidity. Recent experimental researches tend to show that, however rapid the effects, they are still for the most part reconcilable with the view that a poison enters the blood before it begins to manifest its action on the body. It has been stated that poisons are transmitted from the female to the fœtus in utero, and that they are found after death in all parts of the body of the fœtus. This transmission can only take place by the medium of the circulation. (See ante, p. 44.) There is reason to believe that the fœtus is killed by the poison thus circulated with the blood, except in those cases in which death takes place with great rapidity. Mr. Hicks destroyed a pregnant cat by pouring a drachm of prussic acid down its throat. The chest was opened in two minutes after death, and the heart was seen contracting. On laying open the abdomen and examining the uterus, the kittens were found alive, not having apparently suffered from the poison which had destroyed the mother. They continued to move for some time even when removed from the uterine cavity. (Medical Gazette, vol. xxxvi, p. 590.) The blood circulated through the placenta might not have contained a sufficient quantity of the poison to destroy the fœtuses in utero.

The cases which may be adduced against the theory of absorption being necessary to the action of a poison, are so few that they may be regarded as of an exceptional kind; and they will probably disappear by further scientific research.

Remote or systemic action of poisons.—By this we are to understand that power which most poisons possess of affecting some organ or organs remote from the part to which they are applied. The same substance often possesses both a local and remote action: but some poisons affect one organ remotely, and others another. Cantharides, a poison which has a violent local action as an irritant, to whatever part of the body it may be applied, affects remotely the urinary and generative organs. Mercury affects the salivary glands. Morphia, whether applied to a wound or to the mucous membrane of the stomach, affects the brain. Digitalis taken internally affects the heart; strychnia, the upper part of the spinal marrow; prussic acid, the brain and spinal marrow. Belladonna produces a dilatation of the pupils by paralyzing the ciliary nerves, and it produces this effect whether applied locally to the eye or taken into the stomach. MM. Kölliker and Pelikan state that the *Tanghinia*, or poison of Madagascar, has a paralyzing action on the heart and muscular system especially. It paralyzes the nerves; but they regard it as essentially a muscular poison. (Proc. of Royal Society, No. 30, vol.

ix, p. 174.) The woorara poison, when it acts rapidly, destroys life without producing convulsions, and exerts a special paralyzing influence on the nervous system. It acts in a mode precisely the reverse of that of strychnia. The woorara destroys the nervous system from the circumference to the centre, while strychnia, in producing violent convulsions, destroys it from the centre to the circumference. Strychnia acts upon the nerves of motion and sensation. It frequently exalts sensibility to a very high degree. Woorara acts only by paralyzing the nerves of motion, the paralysis of those of sensation being simply a consequence of the asphyxia resulting from the cessation of respiration. But while woorara paralyzes the nerves of motion, it does not destroy the contractility of the muscles; the heart continues to beat in animals poisoned by it. (Bernard, op. cit. 316, 341, 346.) This remarkable poison allows of an entire separation of the two functions of the nervous system,—motion and sensation. In the action of chloroform a converse effect has been noticed: there has been a complete paralysis of sensation, while the nerves of motion have retained their power. Strychnia appears to exert no poisonous action, or but a slight effect, on animals destitute of spinal marrow. Bernard has made this observation on leeches, and I have found that the larvæ of insects may be immersed in a strong solution of strychnia, or even covered with finely powdered acetate of strychnia, without any indication of the effects produced on vertebrated animals by this poison. Aconite has both a local and a remote action. The root when chewed causes a peculiar tingling and numbness of the lips. Its remote action as a result of absorption is manifested chiefly on the nerves of sensation. The late Dr. Pereira found that an alcoholic extract of the root produced complete loss of sensibility in a dog, although the animal was able to walk. (Mat. Med. vol. ii, pt. 2, p. 686.) In some cases, this kind of action is more obscure; and the same poison will affect remote organs differently, according to the form and quantity in which it may have been taken; and perhaps, according to peculiarity of constitution in the poisoned subject. The mineral acids rarely affect the brain remotely; the mental faculties, in cases of poisoning by them, commonly continue clear until the last moment of life. Arsenic sometimes affects the heart,—this is indicated by syncope;—at other times the brain and spinal marrow,—this is known by the coma, stupor, numbness, tingling, and paralysis of the extremities that occasionally supervene in poisoning by this substance. In other cases its effects have been chiefly manifested on the spinal marrow, indicated by violent tetanic convulsions. Oxalic acid was found by Christison and Coindet to affect remotely either the heart, the spinal marrow, or the brain, according to the strength of the solution in which it was administered to animals.

In all cases of *acute* poisoning, *i. e.* cases in which the symptoms run through their course rapidly,—whether the substance have a local action or not, death is commonly referable to the influence exerted by the poison on a remote organ important to life. Most poisons destroy life by affecting the heart, brain, or spinal marrow. The impression produced on either of these important organs is, however, not always so intense as to kill; for individuals have been known to recover from morphia, strychnia, or prussic acid, even after alarming symptoms as a result of this remote influence have manifested themselves. In some instances, however, the impression produced is such as to annihilate, speedily, the vital functions. Thus large doses of hydrocyanic acid, conia, or strychnia may destroy life in a few seconds or minutes, without producing any perceptible local changes on the body. Hydrocyanic acid has in some instances manifested an irritant action on the stomach; but this is by no means a condition necessary to its fatal operation as a poison. If there are some poisons which create difficulty by reason of their rapid action, there are others which appear to require a considerable time for the manifestation of their effects; and here it is not easy to explain why the function of absorption should be suspended. The poison of rabies often lies a long time dormant in the bitten part before it produces hydrophobia in man. Many weeks may elapse before the symptoms appear; although the bitten part has been excised some days after the injury, with the effect apparently of removing the poison, and preventing its absorption into the blood. That there is something of a poisonous nature deposited in the bitten part, appears probable from the fact that it becomes tender and painful shortly before the access of those formidable nervous symptoms which lead to death. Contrast this with the ticunas, the woorara poison, or strychnia, introduced into a similar wound. Here the symptoms are produced in all their intensity in a few seconds or minutes. We know of no poison in which such formidable effects can be so long suspended as in the case of the poison of rabies. That it is absorbed and circulated, and then acts like other poisons, appears probable from the curious fact recorded by Mr. Steele, of its transmission by the milk of the female. This gentleman states that two ewes were bitten by a rabid dog. Rabies appeared in them about six weeks after the bite, and they were killed. One had two lambs, the other one. At first they were permitted to suckle. The lambs were subsequently attacked with rabies, and were then killed. It appears highly probable that they received the poison through the milk, because they were removed from the ewes a month before these became affected: there was no mark of their having been bitten, nor is it proved that a sheep can communicate the poison by a bite, either before or after it has been attacked with rabies. (Med. Gaz. vol. xxv, p. 160.)

Cause of death.—When a poison like concentrated sulphuric acid proves rapidly fatal, without entering the blood by absorption, death is ascribed to the shock impressed on the general nervous system, from the effects of the poison on the living tissues. The nature of the fatal impression thus produced can no more be determined than the nature of thought or sensation. There is, however, no greater difficulty in conceiving that such an impression may be excited by a poison, than that a slight mechanical injury in a remote part of the body may cause an attack of tetanus. (Addison and Morgan on Poisonous Agents, p. 64.) The fact that the greater number of poisons enter the blood and act through this fluid, does not bring us any nearer to an explanation of the direct cause of death. One hypothesis assumes that the organ remotely affected is poisoned by the blood which contains the substance dissolved. This doctrine has been revived by Liebig in a modified form. He considers that an alkaloidal poison—morphia, for example,—may be chemically converted into a substance like brain by the subtraction of some elements, and the addition of others; the quality of the cerebral matter becoming altered, and rendered unfit to support vital energy.

Auglada supposes that a poison when absorbed produces its effects by destroying the vitality of the blood, but all poisons do not act alike; one depresses the action of the heart, another affects the brain, a third the motor tract of the spinal marrow, and a fourth annuls the sensibility of the nervous system generally. The destruction of the vital properties of the blood affords no explanation of these specific differences.

There are few physiologists who doubt that all absorbed poisons act through the blood, and that they alter its physical or its chemical properties, sometimes manifested by an alteration in its consistency, or by a change of color,—the poisonous substance itself simultaneously undergoing a change. This has been established by experiment in reference to oxalic acid, and some other substances (Christison on Poisons, p. 18); and according to the statement of Mialhe, the compounds of arsenic and phosphorus with hydrogen affect the vital properties of blood by the removal of oxygen. As an additional proof, M. Bernard has recently found that cyanide of mercury produces hydrocyanic acid in the capillary system of the lungs (op. cit. p. 66). There is, therefore, reason, from analogy, to believe that organic poisons may undergo some change in this fluid. The fact that a poison can be demonstrated to exist unchanged in the blood and tissues proves nothing to the contrary, because this may be a portion of the undecomposed substance. The question can only be determined by experiment in reference to each poison. Thus, with respect to alcohol, chloroform, and prussic acid, the fact that these liquids can be detected in the blood is consistent with the view that they undergo a partial change. It has been stated that chloroform is circulated and eliminated in an unchanged state, but Dr. Jackson, in examining the blood of a woman who had died from the inhalation of chloroform, found that the chlorine had been replaced by oxygen, and that formic acid existed in the blood, and this was separated from it by distillation. The chlorine had combined with the blood, which had lost the property of coagulation and of becoming red by exposure to air (Chemist, 1856, p. 544). Those, therefore, who have detected chloroform unchanged, have detected merely the surplus. M. Bernard found in a series of experiments that in an animal destroyed by sulphuretted hydrogen, the muscles were dark-colored, while in another killed by the oxide of carbon they were of a bright red. From these opposite conditions of the blood, it might be supposed that carbonic oxide would prove a proper antidote to sulphuretted hydrogen; but an animal placed in a mixture of the gases would die. On the same principle it has been erroneously supposed, that as strychnia is a powerful excitant of the nervous system, a poison like woorara, which depresses or annihilates the nervous power, would be a proper antidote. This mode of treatment is based on pure theory. As Bernard remarks, the convulsions caused by strychnia might be suppressed, but death would take place from such a mixture as certainly and as speedily as if woorara had not been given. Animals, according to him, have died even more rapidly in these experiments than when strychnia or woorara was given separately; although convulsions were suppressed when the two poisons were given at once (op. cit. pp. 53, 377). I agree with him, that this supposition that energetic poisons can thus neutralize the effects of each other is a pure illusion. There is no chemical antidote which can act upon the blood or a poison contained in this liquid. Antidotal treatment, to have any effect, must be directed to that portion of the poison which has not been absorbed or eliminated. The only antidote to the effect of poisons is elimination, or the actual removal of the poison from the system (Bernard, op. cit. 102). That chemical changes of a remarkable kind may take place in the blood and prove fatal by the production of poisons from inert substances, is proved by the following experiment performed by M. Bernard. The emulsine of the sweet almond and the amygdaline of the bitter almond are inert substances; but it is known that when mixed with water or a watery liquid they produce prussic acid. Bernard injected fifteen grains of amygdaline dissolved in water into the jugular vein of a rabbit; no injurious effects resulted. A solution of emulsine produced no effects; but when a quantity of solution of emulsine was injected into the vein soon after the amygdaline,—by the reaction of the two principles in the blood, prussic acid was evolved and the animal died. When the quantity of emulsine was small, although prussic acid no doubt resulted, the animal recovered, the small quantity of the poison formed, having been speedily eliminated by the lungs. The effect depends on the amount of poison produced and circulating at any one time in the blood. These results serve to throw a light on the action of miasmata received into the blood through the lungs, on the action of paludal poisons, the poisons of cholera, of rabies, of serpents, and of the reception of pus or putrid animal matter into the blood by wounds. Such poisons exert no action through the mucous membrane of the stomach or bowels, but if they penetrate into the blood through the thin mucous membrane of the lungs, or by direct contact, they there generate a poison which by its action on the blood affects health or destroys life. Bernard ascribes these changes to an action resembling that of fermentation (op. cit. 96).

It is a curious fact with regard to absorption and elimination, observed by Mitscherlich, Wöhler, and Frerichs, that the essential oil of bitter almonds is oxidized in the system when given in small quantity, and is eliminated in the urine under the form of hippuric acid. When given in large doses, however, they found that a portion of it at least had escaped unchanged into the urine. (Pharmaceutical Journal, vol. xiii, p. 219.) Wöhler found that the red prussiate of potash injected into the blood was eliminated in the urine in the form of yellow prussiate, a fact which showed that the salt had undergone deoxidation (Bernard, op. cit. 90). With respect to alcohol, ether, chloroform, and other narcotics, it is rendered highly probable, as a result of experiments made with these poisonous liquids, that they arrest the processes of oxidation which are carried on in and by the blood, and thus disturb the vital functions.

Admitting that every poison could be chemically detected in the blood, it would yet remain to be explained *how* it operated when there, to destroy life. At present there is no satisfactory theory to account for the fatal effect. All we know from observation is, that the poison when circulating through the bloodvessels destroys life. It may be expected that, in the progress of microscopical and chemical science, the precise effect produced by poisons on the blood will hereafter become a subject of demonstration; but, at present, the *modus operandi* is a perfect mystery. We trace the poison to the circulation, and we observe that death is the result; but neither the chemist nor the microscopist can throw any light upon the changes produced by the poison in the blood or in the organs necessary to life. Some observers have stated that, in arsenical poisoning, the blood is very liquid, as in cases of asphyxia, and that it does not so readily coagulate as in health; but M. Flandin has compared the analysis of healthy blood with that of blood taken from a person poisoned by arsenic, and could perceive no difference in its constitution (Des Poisons, 1, 560). I have frequently had an opportunity of examining by the microscope, the blood of persons poisoned by arsenic, prussic acid, the essential oil of almonds, and other substances, but have not been able to detect any appreciable difference in the form or size of the blood-corpuscles, or any chemical difference in the blood itself. In poisoning by prussic acid it presented in one instance a dark purple color; in poisoning by oxalic acid, it has uniformly presented a dark-brown or olive-green tint; and in poisoning by strychnia, it has been frequently found remarkably liquid and of a very dark color. In spite of these visible effects the microscope has shown no change in the size or form of the corpuscles. In a fatal case of poisoning by strychnia, which occurred to Dr. Ogston, of Aberdeen, the blood-corpuscles (examined some time after death) appeared swollen and their outline irregular, but this may have been a cadaveric change. In another case of death from this poison, the blood drawn while the patient was living, issued from the vein dark-colored and of a tarry consistency, and there were contained in it a number of minute bladders of gaseous matter, showing that it had undergone some chemical changes during life. Bernard found, in his experiments on animals, that the oxide of carbon reddened the venous blood, and that the form of the corpuscles was well preserved for a period of fifteen days; the chemical properties on which the phenomena of respiration depend were changed; the blood preserved its usual vital characters, but it was in reality dead. In causing animals to respire the vapor of prussic acid, a similar change was observed in the venous blood. It acquired a red color, and if the animal died suddenly and its body was examined immediately, the blood was found to be red on both sides of the heart. This change of color is not so persistent, as in the case of death from oxide of carbon (op. cit. p. 194). So striking is this change of color according to Bernard, that he regards the issuing of red blood from a vein as a character of poisoning by prussic acid or carbonic oxide. In one of his experiments M. Bernard endeavored to deoxidize the blood by injecting a solution of pyrogallic acid in a dose of fifteen grains. The dog fell, but partly recovered; the animal died in three or four hours. The pyrogallic acid gave a dark color to the blood as it passed into the lungs; it also gave to it a muddy consistency, but it had not deprived the corpuscles of oxygen. Although much of the acid had passed off by the urine, it had destroyed the globules, dissolved them, and rendered the whole of the blood liquid.

The lungs were of a dark color, almost black, and the blood removed from them contained no globules. Some coagulated blood found in the heart, contained globules; but these disappeared on exposure to air, and the blood became thick and black (op. cit. p. 222). In poisoning by strychnia and some other alkaloids, the blood has been found in a similar state. Is this owing to a deoxidation and dissolution of the globules as a result of the action of the poison?

In animals poisoned by woorara, Bernard found that the blood when examined by the microscope presented no perceptible change. It coagulated when drawn; it was of a dark color in the arteries when respiration was arrested, and the florid color was restored when respiration was re-established (op. cit. p. 306). Fontana found that the serpent-poison darkened the blood and prevented coagulation.

If a poison, such as arsenic, antimony, mercury, or lead, is not immediately thrown out of the body, it may be deposited in the organs without producing any of the effects indicative of poisoning. Bernard thinks that the mineral enters into intimate combination with animal or vegetable matter (op. cit. p. 46); but this is certainly not always the case, since the deposited arsenic or antimony may be partly dissolved out of the tissues by water (ante, p. 70). This experimentalist considers that poisons which are not natural constituents of the blood, are rapidly expelled from that liquid. The metallic substances above mentioned are not normal constituents of human blood. They may be localized or temporarily deposited in the organs, but it is only so long as they are contained in the blood that they operate as poisons. This view is supported by a fact which I have frequently observed, namely, that a small quantity of antimony or arsenic is found in the blood compared with the proportion which has been absorbed and deposited in the organs. It is then that portion of poison which is for the time circulated through the arterial capillary system which produces the effects of poisoning. The deposition in the organs tends, like entire elimination from the body, to suspend their operation on the system. If by any means a poison after deposition is rendered soluble, and again received into and circulated with the blood, symptoms of poisoning may reappear. According to Bernard, some powerful poisons, such as strychnia, nicotina, morphia, and prussic acid, undergo no change in the body; they simply traverse the system, from which they are speedily eliminated without leaving any appreciable trace of their passage; and nevertheless the symptoms which they produce are of a most formidable kind (op. cit. 100). There are again some poisons which are not deposited in the organs, but which are eliminated with great rapidity by the lungs. These are the gaseous poisons, of which sulphuretted hydrogen may be taken as an example. This gas acts as a poison when forming only one eight hundredth part of the atmosphere which is breathed. Bernard injected into the jugular vein of a dog, one-quarter of a cubic inch of a saturated solution of sulphuretted hydrogen in water. The vein was secured above the point of injection to prevent the escape of blood, and the liquid was gently propelled from a syringe towards the heart. A piece of paper soaked in a solution of acetate of lead was placed close to the mouth of the dog. The paper was blackened in from three to five seconds, showing that the gas had been eliminated from the lungs. The elimination was found to be completed in a few seconds, since on suspending the action of the syringe, the lead-paper remained white; but it was again blackened almost immediately on resuming the pressure, whereby more of the poisoned liquid was forced into the blood. The animal sustained no injury from the experiment (op. cit. p. 59). Elimination in reference to this, and probably other gases, may be therefore regarded as almost instantaneous; and this fact explains why persons who are speedily removed from a locality in which sulphuretted hydrogen exists, recover when exposed to pure air, provided the noxious action of the gas on the blood has not been too long continued. A larger quantity of this poison may be introduced into the alimentary canal and eliminated from the lungs without producing symptoms of poisoning; but the absorption and elimination take place more slowly. In another experiment, Bernard injected into the rectum of a dog, two cubic inches of a saturated solution of sulphuretted hydrogen. Lead-paper applied to the mouth of the animal was blackened in sixty-five seconds, and the poison was not entirely eliminated until after five minutes (op. cit. p. 59). This result furnishes an explanation of the well-known fact, that sulphuretted hydrogen, as well as other gases, may be taken with impunity in liquids into the alimentary canal, while the same quantity of the gas introduced into the lungs, would produce immediate death. There is, however, another conclusion which M. Bernard derives from these experiments. The presence of a poison in *venous* blood, provided the quantity is not too large, does not produce the usual effects of poisoning; it may be entirely eliminated before it reaches the arterial capillaries,—the field within which it operates. In the experiment on the alimentary canal, the retardation of the eliminating process is ascribed by him partly to the slow absorption of the gas, and partly to the sluggish circulation through the abdominal venous system (vena portæ).

Isolated facts of this description, however, do not help us to explain how it is that certain organs are specially affected by certain poisons: they point to changes in the blood, but the precise nature of these changes, whether chemical, physical, or vital, as well as the mode in which they affect the brain, spinal marrow, or heart, must be a subject for further experimental inquiry. In the mean time the principal results already obtained may be comprised in the following conclusions.

1. That a large number of poisons susceptible of detection either by chemical analysis, or by their physiological properties, are clearly traceable to the blood.

2. That poisons which act by absorption, are absorbed within a few seconds when placed under circumstances favorable for this process.

3. That unless speedily eliminated from the blood or deposited in the organs, they produce in this fluid physical and chemical changes which lead to death; and that with respect to some of these agents they at the same time partially undergo a chemical change.

4. That elimination by the secretions and deposition in the organs commence so soon as the poison has entered the blood by absorption, and that these processes continue until death, or, in the event of recovery, until all the poison has been thrown out of the body.

5. That the quantity of poison at any one time present in the blood is exceedingly small.

6. That, with a few exceptions among corrosive poisons, there is reason to believe that no poison acts fatally until it has entered the arterial capillary circulation.

CHAPTER VI.

INFLUENCE OF HABIT ON POISONS—OPIUM—STRYCHNIA—ALCOHOL—ETHER—MINERAL POISONS NOT INFLUENCED BY HABIT—ACTION OF ARSENIC ON HORSES AND CATTLE—THE ALLEGED ARSENIC-EATERS OF STYRIA—ARSENIC-EATING A DEFENCE—EXTERNAL APPLICATION OF ARSENIC—TOLERANCE AS IT AFFECTS THE ACTION OF POISONS—OPIUM—TARTAR EMETIC—THE ITALIAN PRACTICE OF CONTRA-STIMULUS—SULPHATE OF COPPER—IDIOSYNCRASY.

Influence of Habit on Poisons.—It is a well-known fact that habit diminishes the effects of certain poisons: thus it is that opium, when frequently taken by a person, loses its narcotic power after a time, and requires to be administered in a much larger dose. Indeed, confirmed opium-eaters have been enabled to take at once a quantity of this drug which would have infallibly killed them, had they commenced with it in the first instance. Even infants and children who are well known to be especially susceptible of the effects of opium, and are liable to be poisoned by very small doses, may, by the influence of habit, be brought to take the drug in very large quantities. This is illustrated by a statement made by Mr. Grainger, in the Report of the Children's Employment Commission. It appears that the system of drugging children with opium in the Factory districts, commences as soon after birth as possible; and the dose is gradually increased until the child takes from fifteen to twenty drops of laudanum at once! This has the effect of throwing it into a lethargic stupor. Healthy children of the same age would be killed by a dose of five drops. This influence of habit is chiefly confined to poisons derived from the organic kingdom. It has been observed that the same influence is manifested in the use of tobacco, alcohol, ether, chloroform, morphia, strychnia, and other alkaloids. The effect of habit on strychnia has narrow limits, and varies with the constitution or the disease under which a person is laboring; hence its use requires to be carefully watched. In a case which occurred to Dr. Booth, of Birmingham, a man, æt. 46, who was affected with hemiplegia, took strychnia in gradually increasing medicinal doses for a period of eleven days without inconvenience. He reached a dose of one grain twice a-day, when the usual effects having ceased to appear, the dose was increased to one grain and a half. He was seized with stupor, loss of speech, and violent tetanic convulsions, and died in less than three hours from the time of taking the poison. (On Poisoning by Strychnia, Guy's Hosp. Reports, Oct. 1856.) In another case a woman, æt. 29, laboring under paralysis, took, in pills, one-sixteenth grain of strychnia daily, and this was increased at intervals of four days, to one-eighth, one-half, and one grain daily. The dose was gradually raised to three grains daily, and this is stated to have been continued for six days! Tetanic convulsions of the limbs and other symptoms then appeared;—the dose was gradually reduced. In two months this patient is said to have taken seventy grains of strychnia. (Med. Gaz. vol. 36, p. 261; Gazette Medicale, Mai, 1845.) The bearing of this large quantity with comparative impunity may have depended not only on habit but on the diseased state of the system (tolerance) and on the rapid elimination of the poison.

The only form in which I have known the question of habit raised in medical jurisprudence is this: whether, while the more prominent effects of the poison are thereby diminished, the insidious or latent effects on the constitution are at the same time counteracted. The answer is of some importance in relation to the subject of life-insurance: for the concealment of the practice of opium-eating by an insured party has already given rise to an action, in which medical evidence on this subject was rendered necessary. As a general principle, we must admit that habit cannot altogether counteract the insidious effects of organic poisons, and that the practice of taking them is liable to give rise to disease or impair the constitution. The habitual use of alcohol may enable a person to take this liquid daily in unusually large quantities, but it does not the less produce disease. The same remark applies equally to the daily use of opium and tobacco. If we believe that these narcotics are absorbed into the blood, and, that until eliminated, they arrest the oxidating processes of this liquid, we must admit that, however such effects may be reduced by habit, so long as they continue in any degree they must undermine health. If it be asserted that the effect of habit is to destroy this action on the blood, and to render the absorbed principles inert, the assertion is without proof or even probability; and in the case of alcohol it is clearly contrary to experience.

Ether, like alcohol, is influenced by habit, and when used medicinally it may after some time be taken with impunity in large doses. Dr. Christison relates the case of a man, æt. 60, laboring under asthma, who, by a gradual increase of dose, was enabled to take every eight or ten days sixteen ounces of ether, and he had continued this practice for many years without any impairment of health. (On Poisons, p. 964.) Dr. Pereira states that a man, laboring under disease of the intestines, took before his death a pint of ether daily to relieve pain. (Mat. Med. vol. ii, pt. 2, p. 467.) In these cases it is probable that the increase of dose is compensated by increased elimination from the lungs and by other channels.

It has been hitherto considered by toxicologists that, except within very narrow limits, habit appears to exercise no influence on the action of *mineral* poisons. There is no proof that a human being has ever accustomed himself by habit to take such substances as arsenic or corrosive sublimate in doses that would prove fatal to the generality of adults. It is well known in the medicinal use of arsenic that a slight increase in the dose has often been attended with such alarming symptoms as to render a discontinuance of the mineral absolutely necessary to the safety of the person. (See ARSENIC, post.)

M. Flandin states that he gave to dogs doses of arsenious acid in powder, commencing with a fraction of a grain mixed with their food; and that in nine months, by gradual increase, they bore a dose of upwards of fifteen grains of arsenious acid, in powder, in twenty-four hours without injury to their appetite or health. (Traité des Poisons, i, 737.) It would appear from this experiment that habit exercises some influence on the action of arsenic in dogs, since the last dose given would have sufficed to kill one of these animals which had not been accustomed to take the poison. We are informed by the late Professor Johnston, of Durham, on the authority of a Dr. Von Tschudi, that horses may not only be made to take large doses of arsenic by habit, but that it renders them fat and plump, gives a bright, glossy skin, and puts them in high health and condition. In fact, the animal, when once used to the arsenic, is said to fall out of condition if it be withdrawn, and is only restored to health by its renewal! (Chemistry of Common Life, vol. ii, p. 205.) It does not appear, however, that he himself had had any experience of this action of arsenic on horses. Mr. Kesteven, who has collected numerous facts on this contested question, has ascertained that the use of arsenic by grooms and carters is a frequent practice in Yorkshire and Suffolk, and there is no doubt that in this attempt to improve the condition of the animal by administering to it a powerful poison, the horse is frequently killed, and death assigned to some other cause. A case, apparently arising out of this practice, was the subject of a trial at the Norfolk Summer Assizes, 1856 (Reg. v. Murton and Petch). The prisoners were charged with the destruction of four horses, by maliciously administering to them a quantity of arsenic. It was clearly proved that the horses died from the effects of arsenic. The defence was that the prisoners, who had the care of the animals, had given arsenic with an innocent intention to improve their coats; and under these circumstances they were acquitted. (Association Medical Journal, September 20, 1856, p. 810.) According to Dr. Von Tschudi, the grooms in Austria give a glossiness to the coats of horses by the use of arsenic in daily doses of three or four grains, but there is an important condition attached to its safe administration, namely that it should be given to the animal only about the time of the new moon! (Journal de Chimie Médicale, 1854, p. 446.) Mr. Kesteven has furnished us with the following statement, made by a farrier of an Austrian cavalry regiment. One grain of arsenic increased to two grains may be given to horses daily mixed with their corn. A fuller appearance is thereby produced, but this arises from a puffiness of the cellular tissue. Such horses sweat with the least exertion. Although the use of arsenic may not be found to injure *healthy* horses *at first*, it eventually proves injurious. Under the administration of the above quantities consumption or wasting away follows sooner or later. If, moreover, great caution be not observed in the employment of such dangerous means, if a certain boundary be overstepped, or a certain order be not observed, symptoms of poisoning are sure to result. (Association Journal, loco citato.) In Cornwall and South Wales the fumes of arsenic from arsenic-works have been found to be very destructive to horses and cattle; the animals have either died with the symptoms of poisoning, or they have lingered some time, have lost their hair, and have suffered from enlargement and disease of the joints. Probably this may be explained by Dr. Von Tschudi as a result of his lunar precautions having been neglected! He tells us that cattle as well as horses may take the poison with impunity, provided due regard be had to the phase of the moon at the time of its administration. (Journal de Chimie Médicale, 1854, loc. cit.) This question respecting the action of arsenic on horses, arose on a trial for murder in which my evidence was required (Reg. v. Geering, Sussex Summer Assizes, 1849). It was suggested that arsenic was a useful and beneficial medicine, and much employed by veterinary practitioners; it was thus attempted in the defence to account for the possession of arsenic in the form of a five-grain pill! An experienced professor at the Veterinary College informs me that, although it is the practice of some English farriers and grooms to give arsenic in small doses, in this country, in order to improve the condition of the skin of these animals, it is in his opinion an unsafe medicine, and its use is generally discountenanced by modern veterinarians. Professor Morton says, "As a therapeutic agent for the horse, arsenious acid can well be dispensed with. It is, however, employed by some as a tonic, in doses of from ten to twenty grains daily, and by others it is used as a vermifuge. When injudiciously administered, death has been the result: the whole of the abdominal viscera have been found in a state of inflammation, and the lining membrane of the stomach and intestines eroded in patches." (Manual of Veterinary Pharmacy, p. 52.) Several cases have occurred to me in which arsenic has proved fatal to the horse, and the appearances above described have been met with. These accidents may be sometimes ascribed to the ignorant employment of arsenic by persons who have been misled by the erroneous views promulgated respecting its safety and utility. There is no reason to believe that horses and cattle, whether English or Austrian, possess any special immunity as a result of habit from the effects of arsenic.

Arsenic-eating.—A graver question, however, arises respecting the alleged influence of habit in counteracting the effects of large and repeated doses of arsenic in human beings. This question affects medical evidence in cases of arsenical poisoning, first, in reference to the motive for the purchase or possession of poison; secondly, in regard to the fatal dose; and thirdly, in regard to its presence in medicines or liquids, or as absorbed arsenic in the dead body. Has habit such an influence as is here assigned to it,—or is the belief in the alleged beneficial effects of arsenic so general that it may reasonably account for the possession of this poison?

Some trials of importance have occurred in which questions connected with the alleged practice of arsenic-eating have been put to medical witnesses.

On the authority of Von Tschudi, we find it stated that in certain parts of Styria and Hungary, there are human beings who take arsenic in doses of two or three grains daily, not only without danger, but with actual benefit to health. Some of these persons who increase the dose incautiously, die from the effects, but others who are more careful and take it according to certain rules, are stated to become robust, to acquire blooming complexions as well as an attractive personal appearance, with a power of ascending heights without fatigue. The practice of eating arsenic, like opium-eating in England, is, we are told, carried on in Styria in secrecy. One peasant, according to Von Tschudi, sixty-three years of age, was in the habit of taking a lump of two or three grains daily, eight or ten times a month, about the period of the *new moon* (!) and he had continued this practice from the age of twenty-seven. In about thirty-five years, we are informed, this man had swallowed from twenty to twenty-two ounces of arsenic. He had, however, always observed the precaution of increasing the dose at the new moon and diminishing it at full moon ! We are further told that there are no symptoms of illness or chronic poisoning produced by this practice in arsenic-eaters, when the dose is properly adjusted to the constitution and habit of body; but we have no information of the mode in which these conditions are discovered. If from any cause the arsenic be discontinued for a time, symptoms occur resembling those of slight arsenical poisoning. (Johnston, op. cit. 204.) While women are said to take arsenic in secret to improve their personal attractions,—men take it to enable them to ascend mountains, to endure fatigue, and to protect them (on homœopathic principles) from the arsenical fumes to which they may be exposed in working the ores of arsenic. (Journal de Chimie Médicale, 1854, p. 445.) Von Tschudi states that in 1852, at some chateau in France, a servant intending to poison his mistress, gave her small doses of arsenic in her food, thinking by these means to conceal the attempt at murder. To the surprise of the criminal the lady improved in health and personal appearance. He then gave a stronger dose,—when the attempt was discovered, and the facts became known. The escape of this lady is referred to the same mode of action of the poison as that which is recorded of the arsenicophagists of Styria ! The locality and date of this alleged occurrence are not given by the writer, and there are no facts to support the strange assumptions which have been built upon Von Tschudi's statements. It is stated that the practice of eating arsenic, in order to be carried out successfully, should be commenced in youth—that when once begun it must be continued, and that symptoms of poisoning by arsenic do not show themselves as in England, when the poison is taken, but only when the use of it is discontinued! Further, there is said to be some risk attending the practice, unless the arsenic be swallowed at or about the period of new moon ! It need hardly be remarked that these absurd and exaggerated statements are utterly inconsistent with all that is known concerning the action of arsenic in this or other countries; and but for the fact that they for a time received the literary support of Professor Johnston, and were diffused by him in an amusing book, they would not have required any serious refutation. Mr. Kesteven has shown that they do not rest upon any trustworthy grounds; and he has collected a number of facts from the arsenic-works of Cornwall, which clearly prove that the so-called arsenic or *hidri* of the Styrian peasants, cannot be arsenic as it is known in England. (Assoc. Med. Jour. 1856, Sept. 6, 757, and Sept. 20th, 810.)

That the peasants of Styria swallow some white powder is not at all improbable. That it is white arsenic has not yet been proved by any analysis. It is probable, from the mode in which the powder is stated to be collected, that it is oxide of zinc; hence Von Tschudi and those who have placed credit in his statements, have overlooked the most important fact in this strange history. It appears, however, according to this writer, that a case has occurred in which this theory was brought forward to account for the presence of arsenic in a dead body. Without giving place, date, or name, we are told that "a few years ago" a remarkable criminal case was tried in his neighborhood. The body of a man had been buried eight years, and was then disinterred for judicial purposes, when on analysis arsenic was found in it. Von Tschudi then says, "The counsel for the prisoner made use óf my communications. I also was called on to give my evidence; and after numerous witnesses had been examined, the conclusion arrived at was almost beyond the possibility of doubt, that the man suspected to be poisoned, was a poison-eater. As the rest of the evidence against the accused was not well founded, both prisoners were acquitted; while, without the knowledge of the strange practice of taking arsenic, a condemnation would most surely have followed. This is but one case among many similar that I could cite." (Association Journal, Sept. 20, 1856, p. 809.) There is no reason to believe that arsenic-eating is practised in this country, still an attempt may be occasionally made to turn this Styrian theory to use for the purposes of a defence. In the case of Reg. v. Wooler (Durham Winter Assizes, 1855), it was actually contemplated by the late Sergeant Wilkins, to account for the unexplained presence of arsenic in the body of Mrs. Wooler, by reference to the opinions of Johnston and Von Tschudi. There was an intention to suggest on the part of the defence, that this lady had for a long period been in the habit of dosing herself with arsenic, unknown to her friends, for the purpose of improving her personal appearance in the eyes of her husband; that her body had become habituated to it, and that in fact she had died only because she had latterly left off the practice; but it was prudently abandoned, on the principle that a bad or inadequate explanation is worse than none. According to the evidence given in this case, symptoms of poisoning by arsenic first showed themselves about six weeks before she died; they occurred at intervals with aggravation during this period. She had no access to arsenic in any shape in the six weeks preceding her death. When she died, arsenic was found in all parts of her body,—the result of absorption and deposition. The poison was also eliminated in the urine up to within a few days of her death. Hence, to explain some of the facts on the arsenic-eating theory, it would have been necessary to assume, contrary to the evidence furnished by the urine, that this poison is not eliminated from the tissues.

The published accounts of alleged arsenic-eating lead to the inference that symptoms of poisoning are not caused by the use of the poison, but as a result of its withdrawal. This is quite contrary to all experience regarding the action of arsenic and other poisons in this country. The symptoms are observed only where a poisonous substance is administered, and cease when it is withdrawn or eliminated from the body. Hence no medico-legal inquiry is likely to arise regarding this alleged practice, which can affect medical evidence any more than the practice of opium-eating. If the dose of arsenic or opium is small, there are presumed to be no ill effects; if large, there will be symptoms of poisoning and death. Should arsenic be found in a dead body in small quantity, and there are no appearances indicative of recent administration, the discovery could not embarrass medical evidence, because arsenic is largely used as a medicine; and unless symptoms of poisoning have manifested themselves during life, and there are appearances in the body indicative of its action, there can be no ground for alleging that a person has died from its effects. If, however, such symptoms and appearances are met with, and the poison is found in the dead body, then the inference will be, that the death of the deceased, whether an arsenicophagist or not, was caused by arsenic. The case will then resolve itself into one of accident from over-dose, suicide, or murder; and, as in Reg. v. Wooler, unless it can be proved clearly and conclusively, that some one administered the poison, a charge of murder cannot be sustained. It must rest with the moral evidence to show whether death from arsenic was the result of accident or suicide. Assuming that one-half of the statements is based on truth, they prove,—not that arsenic may be swallowed in very large and increasing doses as an effect of habit,—for it is said that the same dose (two or three grains) was taken for many years,—but that there must be some national idiosyncrasy among the Styrians which renders them proof against the effects of poisonous doses of arsenic under certain circumstances.

Mr. Hunt, who has probably used arsenic medicinally to a greater extent than any practitioner in this country, states that its beneficial or curative powers reside alone in doses too small to produce the ordinary effects of poisoning. Further, habit appears to have so little influence over its use, that the safe plan of administration is the reverse of that of other medicines, such as opium and antimony. A full dose of arsenic should be given at once, and the quantity gradually decreased. By a full dose, this writer implies about the eighth part of a grain, taken in three doses in one day. He has found that extreme doses, such as those which are alleged to be taken by the Styrians, produce so long and lasting an impression on the nervous system, especially in delicate subjects, as to render it for months or years subsequently, so intolerant of the medicine, as absolutely to interdict its use. (Pathology of Diseases of the Skin, 1847, p. 13.)

A most extraordinary use for the purposes of a defence was made of this Styrian doctrine at the trial of Miss Madeline Smith (Edinburgh Court of Justiciary, July, 1857), for the murder of L'Angelier. To account for the purchase of arsenic, the accused stated that she had used it as a *cosmetic*. The deceased died evidently from the effects of arsenic, on the 23d of March. Irrespective of two previous purchases of colored arsenic, for which false reasons had been assigned, it was proved that the prisoner had purchased one ounce, as she said, "to kill rats," on the 18th of March, only five days before the death of the deceased. The arsenic was sold colored with indigo. When charged with the crime, and required to account for the poison, she stated that she had bought arsenic on various occasions; that she had used it all as a cosmetic, and had applied it to her face, neck, and arms, diluted with water; that a companion at school had told her that arsenic was good for the complexion. This was directly contradicted by the person whom she had named as her informant; it was proved that she had left school in 1853, and that her purchases of arsenic for cosmetic purposes had only commenced four years afterwards, in February, 1857, *i. e.* during her secret intimacy with the deceased. It was urged, that this mode of using arsenic externally had never been suggested in any popular publication. In fact, Von Tschudi has not recommended the use of the mineral for washing the face; and an ounce of arsenic colored with indigo, could scarcely be expected to improve the complexion. This, however, it was suggested, might have arisen from ignorance or mistake on the part of the accused, respecting the precise mode of using it. To support this theory, Dr. Laurie was called, and he deposed that he had washed his hands and face in water containing a quantity of arsenic colored with indigo, and he had found no disagreeable effects from it. Soon afterwards, however, he washed his face with cold water, and he stated that he would not advise the external use of arsenic as a practice.

It is hardly a question of science, but one of common sense, whether a woman of adult age would use an ounce of arsenic colored with indigo or soot in the manner and for the purposes suggested. A physician knowing the properties of arsenic, would take care to keep the poison out of his eyes, nose, and mouth, and relieve himself of risk by speedy ablution afterwards. It is to be hoped that the evidence of this physician as to the immunity which he experienced, will not induce others to improve upon Von Tschudi's practice, and freely use arsenic externally as well as internally for benefiting the complexion.

So far as habit is concerned, it may be said that neither the internal nor external use of arsenic can be resorted to with safety. Of the danger arising from external application, Mr. Kesteven has already furnished some examples (Association Journal, Sept. 20, 1856, p. 811); and it is only the integrity of the skin which can save a man from serious accidents when arsenic is used for washing the face and hands in the mode in which it was used by the medical witness, and professed to have been used by the prisoner, Madeline Smith. The contact with the mucous membrane in any part, or an open wound, would lead to serious consequences. It has been found that even the accumulation of small particles of arsenic in the depressions or in the hollows of the unbroken skin, is attended with some risk. (Association Journal, ut supra.)

Tolerance.—There are certain conditions of the body in which, without reference to habit, a large dose of a poisonous substance may be taken at once by a person who may not have previously taken it as a medicine, and yet the ordinary effects of poisoning will not be manifested. In tetanus and hydrophobia, poisonous doses of opium have been given at short intervals without producing any injurious symptoms. In a case of tetanus, which occurred while I was a student at Guy's Hospital, one ounce of tincture of laudanum—a quantity sufficient to destroy four persons—was given to the patient every four hours without producing any of the symptoms of poisoning. In fact, in prescribing this drug for the disease, surgeons look rather to the effect produced, than the actual quantity taken by the patient. Mr. Curling mentions a case in which four ounces of laudanum were given daily for eleven days, without affecting the progress of the disease, or producing narcotism. (Curling on Tetanus, p. 152.) Infants, it is well known, are peculiarly susceptible of the effects of opium, so that they are sometimes destroyed by doses of one or two minims. On the other hand, in certain forms of disease,—like adults they will take large doses of this drug without injury. Dr. Nevins has reported the case of a negro child, aged fifteen months, affected with idiopathic tetanus, to which two minims of laudanum and four of spirit of sulphuric ether were given every hour. The next day the rigidity of the muscular system was undiminished, although the child had taken about *forty minims* of the tincture. Four minims of laudanum were then given every hour, and on the following day the rigidity had much abated. The child had then taken *two drachms* of laudanum in two days, but there were no signs of narcotism. (Medical Gazette, vol. xlvi, p. 981.)

Tartar emetic presents a similar peculiarity. This medicine has been safely and beneficially prescribed in large doses and for a long continuance in pulmonary diseases and rheumatism. Tommasini and Laennec were in the habit of prescribing largely on the Italian theory of contrastimulus. Persons affected with pulmonary disease manifested, generally speaking, a "tolerance" of the medicine if given in large doses and at short intervals. When this tolerance was once set up, the medicine was productive of benefit, but when not established, either from peculiarity of constitution or other causes, it was withdrawn. (See Pereira, Materia Medica, 4th ed. vol. i, p. 101; Forbes's Translation of Laennec on Diseases of the Chest, pp. 251, 260; also Della Nuova Dottrina Medica Italiana, del Prof. Giacomo Tommasini, Firenze, 1817.) The facts connected with the tolerance of certain medicines in poisonous doses, are of some medico-legal importance. Although well known to professional men, it is a remarkable circumstance that they should have been recently adduced by learned physicians as furnishing a proof that tartar emetic is not a poison, and is not likely to destroy life! When the death of Ann Palmer was referred by Dr. Rees and myself to the repeated doses of tartar emetic which had been administered to her, the practice of Tommasini in giving large doses with safety, was adduced by certain medical writers as proving the very reverse of what that eminent physician intended. Tommasini and Laennec did not prescribe large doses of this mineral for persons whose state of health did not require antimony in any shape; and they gave especial precautions respecting the use of it in diseases of the lungs,—that where irritant effects followed, it should be withdrawn; and it could not then be persisted in without endangering the life of a patient. As well might these writers have argued that opium was not a dangerous drug, and might be given to a healthy person in large or frequent doses, because they had known such doses of it borne without any symptom of poisoning in cases of tetanus and hydrophobia! The absurdity of such reasoning would have been at once perceived had Ann Palmer been treated with such heroic doses of opium as are safely given in tetanus,—but it was entirely overlooked with respect to the use of tartar emetic; and Tommasini was thus incorrectly made responsible for justifying a new system of murder by poison in this country. The fruits of these injudicious publications were seen in the trial and conviction of a woman (McMullen), at the Liverpool Autumn Assizes, 1856, for the murder of her husband, by small doses of this substance, and of a man, named Hardman, for the murder of his wife by similar means (Reg. *v*. Hardman, Liverpool Autumn Assizes, 1857). An error like this, once widely circulated, is not easily eradicated.

The authority of Orfila has been erroneously quoted in support of the view that large doses of antimony might be given with comparative impunity. This distinguished medical jurist, in speaking of the doses of poisonous substances, makes the following remarks: "Persons laboring under certain forms of disease, will bear without injury, considerable doses of a poisonous substance, while much smaller doses would produce dangerous effects on the same persons in a *normal state*. We may cite, in proof of this, the effects of *tartar emetic* in inflammation of the lungs, etc. Would anybody venture to assert that these poisonous substances are not deleterious to man, because they do not always act as poisons, even in very large doses? Certainly not; it would merely prove that those substances, which are really poisonous in the generality of cases, are not poisonous in the same doses under certain conditions in which they are *tolerated*." (Toxicologie, 5ème edition, 1852, tome i, page 18.) That barristers, in defending an accused person, should quote only such parts of a book as precisely suited their purpose, and endeavor to suppress all others, is not surprising; but that medical writers pretending to instruct the profession, should thus deal with an authority with a view of condemning by anticipation, opinions solemnly expressed on oath by witnesses for the Crown, is, to say the least, an unwarrantable practice. The only excuse for such conduct is, that they had not read the book from which they quoted.

This tolerance of poisonous medicines in large doses may also account for certain facts, noticed by Dr. Hœnerkopf, on the comparative impunity with which *sulphate of copper* may be taken by children affected with croup, in the treatment of which disease it appears to be much used by German physicians. Dr. Hœnerkopf gave to a child affected with croup, 216 grains of sulphate of copper in eight days, making a daily average of twenty-seven grains. To another child, four and a half years old, he gave 150 grains in seven days; to a third, two years old, he gave 189 grains in twenty-four days, or about eight grains daily; and he cites other cases in which equally large doses were given, not only with impunity, so far as symptoms of poisoning were concerned, but with benefit to the patient. (See Casper, Vierteljahrschrift, 1855, p. 227.) I quite agree with Dr. Spielman, who, in commenting upon these cases, observes that, from such data, it would be most illogical to contend that sulphate of copper was not a poison; and that it might be given to healthy persons in similar doses with impunity. (Casper's Vierteljahrschrift, 1856, ii, p. 45.) Habit may, to a certain extent, allow of a slight increase or frequent repetition of the dose; but there is reason to believe that the powers of the medicine are spent on the disease, and it is only on recovery that these large doses, if continued, exert a poisonous action. The noxious action of this substance is suspended. This is clearly the case with opium and morphia in the treatment of mania, tetanus, and hydrophobia. (See post, p. 109.) Dr. Rees informed me of a case in June, 1857, in which a lady suffering from acute puerperal mania, took thirty grains of hydrochlorate of morphia in twenty-four hours, without any injurious consequences. She had not been accustomed to take narcotics. Dr. Rees sent me a portion of the morphia-salt, which I found to be of the usual pharmaceutical kind. This was an instance of tolerance from disease.

A case, of which the details are elsewhere given (ante, p. 93), appears to indicate that, in certain forms of paralysis, there may be a tolerance of *strychnia* in large and fatal doses, although, undoubtedly, habit in this instance influenced materially the operation of this poison.

Idiosyncrasy.—This is a term applied to that peculiar condition of body, the reverse of habit, in which small medicinal doses of poisons, such as opium, arsenic, strychnia, mercury, or antimony, so seriously affect a person as to endanger life. Thus it increases the effect of poisons, or confers on an ordinary medicinal dose a poisonous instead of a curative action. On other occasions, irrespective of habit or of tolerance from disease, a large dose of a poison may be taken and produce no dangerous consequences. Dr. Christison mentions a remarkable instance of this kind, in which a gentleman unaccustomed to the use of opium took nearly an ounce of good laudanum without any effect. (On Poisons, p. 32.) This form of idiosyncrasy, by which poisons cease to operate as such, is comparatively rare; for, as a general rule, no particular state of the body is a safeguard against their operation; but daily experience teaches us, that some persons are more powerfully affected than others by an ordinary dose of opium, arsenic, antimony, and other substances. Some persons cannot tolerate arsenic in any dose; some are readily affected with lead-disease from causes from which the greater number of persons do not suffer. There are others on whom small medicinal doses of arsenic, mercury, antimony, or opium, produce serious symptoms. I have known the twenty-fourth part of a grain of tartar emetic to produce in an adult, nausea, vomiting, and extreme depression. A third form of idiosyncrasy is seen, where a substance, generally reputed harmless and used as an article of food, produces effects so closely resembling those of poisoning, as very frequently to have given rise to serious mistakes. This is the case with pork, certain kinds of shell-fish, and mushrooms. There may be nothing poisonous in the food itself; but it acts as a poison in particular constitutions; whether from its being in these cases a poison *per se*, or rendered so during the process of digestion, it is difficult to say.

The subject of idiosyncrasy is of some importance in a medico-legal view, when symptoms resembling those of poisoning follow a meal consisting of a particular kind of food. In such a case, without a knowledge of this peculiar condition, we might hastily attribute to poison, effects which were really due to another cause. It would appear that in some instances idiosyncrasy may be acquired—*i. e.* a person who, at one period of his life, had been in the habit of partaking of a particular kind of food, may find at another period that it will disagree with him. If pork has been disused as an article of diet for many years, it cannot always be resumed by individuals with impunity. When the powers of life have become enfeebled by age, the susceptibility of the system to poisons is increased; thus aged persons may be killed by comparatively small doses of arsenic and opium. Opium especially affects the aged who are laboring under pulmonary disease. Infants have been destroyed by very small doses of opium. Cases of acquired idiosyncrasy are very rare; it appears to be, if we may so apply the term, a congenital condition.

CHAPTER VII.

CLASSIFICATION OF POISONS—SPECIAL CHARACTERS OF IRRITANTS—DIFFERENCE BETWEEN CORROSIVE AND IRRITANT POISONS—NEUROTIC POISONS—CEREBRAL, SPINAL, AND CEREBRO-SPINAL POISONS.

POISONS were formerly arranged in three classes according to the kingdom from which they were obtained; and thus we had mineral, animal, and vegetable poisons. The inutility of such a classification must be apparent when it is considered, that we do not, by adopting it, acquire any knowledge of the properties of a poison or of its action on the economy. If applied at all, it should be only in a form subordinate to a physiological classification, so as to allow of the arrangement of poisons in analogous groups. A recent writer on Toxicology, M. Flandin, has endeavored to revive the old division of poisons into mineral, vegetable, and animal. (Traité des Poisons, i, 225, ed. 1846.) There appears to be no good reason for the re-introduction of this classification, while there are many objections to it. In stating that opium is a narcotic, or that cantharides is an irritant poison, we convey some idea of the mode of action of these substances; but it is not so when we apply to them only the terms vegetable and animal. We are then left in entire uncertainty as to their mode of operation. All classifications are necessarily more or less arbitrary; but in making our selection, we are bound to prefer that which, while it arranges poisons in a certain order, carries us beyond the mere knowledge of the kingdoms from which they are derived.

In the latest edition of his work, Orfila has divided poisons into four classes:—1. Irritants; 2. Narcotics; 3. Narcotico-acrids; and 4. Septic or putrefying poisons. The first class included by far the largest number of bodies, namely, all mineral poisons, and many belonging to the vegetable kingdom;—while the second class contained substantially only two,—opium and prussic acid. The fourth class included the various animal poisons with a few gases, and the remaining substances were mixed indiscriminately, under the designation of narcotico-acrids. This classification, in a modified form, has been generally adopted by English writers: it was followed in the former edition of this book. M. Galtier, writing in 1855, divides the subject into Inorganic and Organic poisons, the former class including, 1. Metalloidal; 2. Acid; 3. Alkaline; and 4. Metallic poisons; while in the latter class are placed, 1. Vegetable poisons; 2. Animal; 3. Alimentary substances (mushrooms, unwholesome food, &c.); 4. Gaseous poisons. While poisons do not admit of a perfect arrangement, either according to their effects—the organs which are affected by them—or the kingdom of nature from which they are derived, there is room for the adoption of a modification of these arrangements, which, although not free from objection, appears to me sufficient for practical purposes.

The substances called IRRITANT poisons, are so well marked in their characters, that they are retained as a class, divisible into three sections, according to their nature, namely MINERAL, VEGETABLE, and ANIMAL; and the mineral or inorganic irritants are again divided into the subsections *Non-metallic* and *Metallic poisons.* Those irritants which are derived from the vegetable and animal kingdoms, except savin and cantharides, are not very often employed criminally.

Considering the GASEOUS poisons sufficiently peculiar in their properties and effects on the body to require a separate classification, the remaining substances belonging to the vegetable kingdom may be placed in one large class under the name of NEUROTIC poisons. Their effects are chiefly manifested on the brain, spinal marrow, and nerves; some more particularly affect the brain, others the spinal marrow, and others again both of these organs. In the vegetable state, as in the form of roots, leaves, or seeds, they may give rise to pain and irritation in the alimentary canal, but the active principle, when separated from the plant, does not commonly produce these effects, except when it is of an acrid or of a corrosive nature.

The class of NEUROTICS, here constructed out of the Narcotic and Narcotico-irritant classes, admits of a division into three sections, according to the organ especially affected by the poison, namely, CEREBRAL, SPINAL, and CEREBRO-SPINAL.

IRRITANTS	MINERAL	NON-METALLIC	Acids. Alkalies and their salts. Metalloids.
		METALLIC	(*Arsenic.*)
	VEGETABLE	(*Savin.*)	
	ANIMAL	(*Cantharides.*)	
NEUROTICS	CEREBRAL	(*Morphia.*)	
	SPINAL	(*Strychnia.*)	
	CEREBRO-SPINAL	(*Conia, Aconitina.*)	

The *Cerebral* poisons include the pure narcotics, such as opium, with its alkaloid morphia, hyoscyamus, and a few other substances. Their action is, as the name is intended to imply, chiefly confined to the *brain.* They produce stupor and insensibility without convulsions. The *Spinal* poisons are those, the action of which is chiefly confined to the spinal marrow, manifested by violent convulsions, sometimes of the tetanic, and at others of the clonic kind. Spinal poisons do not necessarily cause a loss of sensibility or consciousness: there is rarely any symptom of narcotism when they are taken or administered as poisons. Nux vomica and its alkaloid strychnia are types of this form of poisoning. The *Cerebro-spinal* poisons include those which produce symptoms indicative of an action on the brain and spinal marrow, delirium, convulsions, coma, and paralysis. Conia, aconitina, and atropia, as well as the plants from which they are derived, furnish examples of this group of poisons. In some instances, their effects are specially manifested on the nerves of motion, by exciting or paralyzing them, in other instances on the nerves of sensation by exalting or destroying sensibility; but in the greater number of cases effects are produced on both. In a few instances, a poison appears to affect directly the contractility of the muscles, irrespective of its action on the nerves.

IRRITANT POISONS.—The *irritants* are possessed of these common characters. When taken in ordinary doses they occasion speedily violent vomiting and purging. These symptoms are either preceded, accompanied, or followed by intense pain in the abdomen, commencing in the region of the stomach. The peculiar effects of the poison are chiefly manifested on the stomach and intestines, which, as their name implies, they irritate and inflame. Many substances belonging to this class of poisons possess corrosive properties, such as the strong mineral acids, caustic alkalies, bromine, corrosive sublimate, and others. These, in the act of swallowing, are commonly accompanied by an acrid or burning taste, extending from the mouth down the œsophagus to the stomach. Some irritants do not possess any corrosive action,—of which we have examples in arsenic, the poisonous salts of baryta, carbonate of lead, cantharides, &c., and these are often called pure irritants. They exert no chemical action on the tissues with which they come in contact; they simply irritate and inflame them.

Difference between corrosive and irritant poisons.—There is this difference between CORROSIVE and IRRITANT poisons. Under the action of corrosive poisons, the symptoms are commonly manifested immediately, because mere contact produces destruction of a part, usually indicated by some well-marked symptoms. In the action of the purely irritant poisons, the symptoms are generally more slowly manifested, rarely showing themselves until at least half an hour has elapsed from the time of swallowing the substance. Of course, there are exceptions to this remark; for sometimes irritants act speedily, though seldom with the rapidity of corrosive poisons. It is important, in a practical view, to distinguish whether in an unknown case, the poison which a person, requiring immediate treatment, may have swallowed, is of an irritant or corrosive nature. This may be generally determined by a knowledge of the time at which the symptoms first appeared after the suspected substance was taken. In this way we may often easily distinguish between a case of poisoning from arsenic and one from corrosive sublimate. There is also another point which may be noticed. As the corrosion is due to a decided chemical action, so an examination of the mouth and fauces may enable us to determine the nature of the poison swallowed.

It has been already stated that there are some irritant poisons which have no corrosive properties, and therefore never act as corrosives; but it must be remembered that every corrosive may act as an irritant.

Thus the action of corrosive sublimate is that of an irritant poison, as while it destroys some parts of the coats of the stomach and intestines, it irritates and inflames others. So again most corrosive poisons may lose their corrosive properties by dilution with water, and then they act simply as irritants. This is the case with the mineral acids and bromine. In some instances, it is not easy to say whether an irritant poison possesses corrosive properties or not. Thus oxalic acid acts immediately, and blanches and softens the mucous membrane of the mouth and fauces, but I have not met with any decided marks of what could be called chemical corrosion produced by it in the stomach or viscera.

NEUROTIC POISONS.—Neurotic poisons act chiefly on the brain and spinal marrow. Either immediately or some time after the poison has been swallowed, the patient suffers from headache, giddiness, paralysis, stupor, delirium, insensibility, and, in some instances, convulsions. The cerebral poisons, or those which affect the brain only, have no acrid burning taste like the corrosive irritants; and they rarely give rise to vomiting or diarrhœa. When these symptoms follow the introduction of the poison into the stomach, the effect may be ascribed either to the quantity in which the poison has been taken and the mechanical distension of the stomach thereby produced, or to the poison being combined with some irritating substance, such as alcohol. The purely cerebral and spinal poisons are not found to irritate or inflame the viscera.

Notwithstanding the well-defined boundary thus apparently existing between these two classes of poisons, it must not be supposed that each class of bodies will always act in the manner indicated. Some irritants have been observed to affect the brain or the spinal marrow remotely, *i. e.* through the circulation, and as the result of absorption. This is the case with oxalic acid and arsenic. Both of these common poisons have in some instances, from the first, given rise to symptoms closely resembling those of narcotic poisoning; namely, coma, paralysis, and tetanic convulsions. In a case of poisoning by arsenic which occurred to Dr. Morehead, of Bombay, the symptoms of narcotism were so strongly marked, that it was believed at first the man had taken a narcotic. (Med. Gaz. vol. xliii, p. 1055.) I have met with one case of poisoning by arsenic in which there was paralysis of the extremities, with an entire absence of purging, during the eight days which the deceased survived. In fact, there is in some cases a nearly complete substitution of one set of symptoms for another. An intelligent writer has assumed that these unusual effects of irritant poisons are only observed in the final stage, *i. e.* immediately preceding death; and as these effects are similar in many cases, though produced by different agents, he considers it to be an error on the part of toxicologists, to apply the term narcotic to the effects produced by oxalic acid or arsenic (Billing's Principles of Medicine, 107). The case by Dr. Morehead above quoted, shows among numerous other examples, that narcotic symptoms may be produced primarily by arsenic, and not merely as a secondary result, from exhaustion of the vital powers in the last stage of poisoning. On the other hand, in the chapter on opium, a case of poisoning by a large dose of this drug will be found related, in which there was an absence of the usual symptoms of cerebral disturbance, and the presence of others resembling those of irritant poisoning—namely, pain and vomiting. Thus, then, we must not allow ourselves to be deceived by the idea that the symptoms are always clearly indicative of the kind of poison taken. The narcotic poisons are few in number, and belong to the vegetable kingdom. Some of the poisonous gases possess a narcotic action.

Some poisons belonging to this class, when taken in the form of leaves or roots, have a compound action. At variable periods after being swallowed, they give rise to pain, vomiting, and sometimes diarrhœa; like irritants, they sooner or later produce delirium, stupor, coma, paralysis, and convulsions, owing to their effect on the brain and spinal marrow; but they vary much in their mode of operation. They possess the property, like irritants, of irritating and inflaming the stomach and bowels. As familiar examples, we may point to monkshood, tobacco, belladonna, and poisonous mushrooms. This section of poisons (cerebro-spinal) is very numerous, embracing a large variety of well-known vegetable substances; but they rarely form a subject of difficulty to a medical practitioner. The fact of the symptoms occurring after a meal at which some suspicious vegetables have been eaten, coupled with the nature of the symptoms themselves, will commonly indicate the class to which the poison belongs. Some neurotic poisons have a hot acrid taste, such as aconite or monkshood, while others are intensely bitter, such as picrotoxia, strychnia, brucia, and morphia.

The greater number of poisons belong to the class of irritants and to the cerebro-spinal subdivision of the class of neurotics. It is, in fact, rare to find that the brain is affected without the spinal marrow, or *vice versâ*. Hence the number of poisonous substances, which can be truly called cerebral or spinal, are very few.

Among the poisonous gases some act as irritants on the throat and lungs (ammonia and nitrous acid); others act on the brain chiefly (carbonic acid and carbonic oxide), they are cerebral poisons; while others again produce their effects on the brain and spinal marrow, causing coma and convulsions (sulphuretted hydrogen and cyanogen).

We are at present hardly acquainted with the special action of some of the substances enumerated and classified as poisons; they have been arranged in this work according to their effects as ascertained by toxicologists from experiments on animals, as well as from the few cases in which they have acted as poisons in the human subject.

CHAPTER VIII.

EVIDENCE OF POISONING IN THE LIVING SUBJECT—SYMPTOMS OCCUR SUDDENLY—CAUSES OF RETARDATION OF SYMPTOMS—ACTION OF POISONS AGGRAVATED BY DISEASE—SYMPTOMS CONNECTED WITH FOOD OR MEDICINE—SUDDEN DEATH FROM NATURAL CAUSES MISTAKEN FOR POISONING—SEVERAL PERSONS ATTACKED SIMULTANEOUSLY—EVIDENCE FROM THE DETECTION OF POISON IN FOOD.

We may now proceed to consider the evidence of poisoning in the living subject. To the practitioner the diagnosis of a case of poisoning is of great importance, as by mistaking the symptoms produced by a poison for those arising from natural disease, he may omit to employ the remedial measures which have been found efficacious in counteracting its effects, and thus lead to the certain death of a patient. To a medical jurist a correct knowledge of the symptoms furnishes the chief evidence of poisoning, in those cases in which persons are charged with the criminal administration of poison with intent to murder, but from the effects of which the patient ultimately recovers. The symptoms produced during life, constitute also an important part of evidence, in those instances in which the poison proves fatal. At present, however, we will suppose the case to be, that poison has been taken and the patient survives. Most toxicological writers have laid down certain characters whereby it is said symptoms of poisoning may be distinguished from those of disease.

1. IN POISONING, THE SYMPTOMS APPEAR SUDDENLY, WHILE THE PERSON IS IN HEALTH.—It is the common character of most poisons, when taken in the large doses in which they are usually administered with criminal intent, to produce serious symptoms either immediately or within a very short period after they have been swallowed. Their operation, under such circumstances, cannot be suspended, and then manifest itself after an indefinite interval; although this was formerly a matter of universal belief, and gave rise to many absurd accounts of what was termed *slow* poisoning. In modern times, the negroes of Martinique have been said to possess this art, but the researches of Dr. Rufz show that this is an erroneous statement. (Annales d'Hygiène, 1844, i, 392; also ii, 170.) It is very true that these powerful agents, given at intervals in small doses, do not cause those striking symptoms upon which a practitioner commonly relies as evidence of poisoning. They may then produce disorder, but of so slight a nature, as scarcely to excite suspicion. In fact, under these circumstances, the symptoms often so closely resemble those of disease, that an experienced practitioner may be easily mistaken respecting their origin, especially when no circumstances exist to create the least suspicion of criminality on the part of relatives and others around the patient. Arsenic given in small doses, at long intervals, has thus occasioned symptoms resembling those which depend on chronic disease of the stomach. After repeated attacks and recoveries suspicion may be completely disarmed. Among several cases of this kind which have been referred to me for investigation, was one in which it was alleged that a farmer in one of the midland counties had been poisoned two years before by his housekeeper, who was a respectable person, and most attentive to him as a nurse during his illness. He had been attacked at intervals with vomiting and other signs of disorder of the stomach about three months before his death, but recovered under medical treatment. About eight days before his death the symptoms recurred with greater violence than ever, and he sank under them. They were referred to ulceration of the stomach, so closely did they resemble those of disease. As there was no suspicion of poison, the body was not examined; and nothing would have been known respecting the real cause of death, but for a statement made two years afterwards, by the housekeeper, that she had on two occasions administered to her master

small doses of arsenic, and the last, probably from its being larger than the first, had occasioned death. In the case of Reg. v. Wooler (Durham Winter Assizes, 1855), it was proved that the deceased had been laboring under symptoms of poisoning by arsenic, for a period of about six weeks before her death. The symptoms showed that she must have received the poison at different periods in small doses. At first they were referred to disease. It was, however, their continuance and their occasional violent recurrence in spite of treatment, that induced a suspicion of poisoning, which was confirmed by a chemical examination of the urine, and subsequently of the body. This is the only form of slow poisoning now known to toxicologists. Again, there are what are called *accumulative* poisons,—substances which, in small divided doses, given at long intervals, produce scarcely any perceptible effect on the system; but which appear to accumulate in the body, and their power is said to be unexpectedly manifested with sudden and violent energy. To these forms of poisoning, which it is extremely rare to meet with on criminal charges, the characters about to be described are not applicable.

When poison is criminally administered, it is almost always in such doses as to cause the symptoms to appear *suddenly*, and to run their course with great rapidity. The symptoms of poisoning by prussic acid, oxalic acid, or the salts of strychnia, generally appear either immediately or within a very few minutes after the poison has been swallowed. In one case, however, where the dose of prussic acid was small and insufficient to produce death, the poison was supposed by the patient not to have begun to act until after the lapse of fifteen minutes. (Ed. Med. and Surg. Journal, vol. lxix, p. 72.) The symptoms caused by arsenic and other irritants, and, indeed, by all poisons generally, are commonly manifested in from half an hour to an hour. It is rare that the appearance of the symptoms is protracted for two hours, except under certain peculiar states of the system. It is said, that some neurotic poisons, such as the poisonous mushrooms, may remain in the stomach twelve or twenty-four hours without giving rise to symptoms; and this is also affirmed to be the case with some animal irritants, such as decayed meat; but with regard to the first point, it has been shown by Dr. Peddie, that mushrooms have produced symptoms in half an hour; and a case has fallen under my own observation, in which the symptoms from noxious food came on within as short a time after a meal, as is commonly observed in irritant poisoning by mineral substances. In cases of poisoning by phosphorus, the symptoms do not commonly begin until after the lapse of some hours.

Influence of sleep.—The symptoms produced by some of the more common poisons, are apt to be retarded under certain conditions of the system. When an irritant poison is taken on a full stomach, the symptoms do not usually appear so speedily as when the stomach is empty. So again, it is stated by Dr. Christison, from cases which have fallen under his notice, that *sleep* retards the action of arsenic, and the same may hold with other irritants. Thus, if a person should happen to fall asleep soon after swallowing a poison, it may not produce the usual symptoms until four or five hours afterwards, or the occurrence of these may be even longer protracted. This is supposed to be owing to the general state of insensibility of the body, and the depressed condition of the nervous system during sleep.

Influence of intoxication.—This state has been considered to retard the operation of opium. Observations of this kind must, of course, be accidental; and there is scarcely a sufficient number of cases reported of narcotic poisoning under these circumstances, to justify a decided opinion on the point. It was observed of a person who had swallowed a strong dose of opium, while partially intoxicated, that the symptoms were some hours before they were manifested. Perhaps, strictly speaking, the symptoms in these cases are masked.

Influence of disease.—A *diseased* state of the body may render a person comparatively unsusceptible of the action of some poisons, while in other instances it may increase their action, and render them fatal in small doses. In dysentery and tetanus, a person will take, without being materially affected, a quantity of opium sufficient to kill an adult in average health. Mania, cholera, hysteria, and delirium tremens, are also diseases in which large doses of opium may be borne with comparative impunity. In a case of hemiplegia, a woman æt. 29, took for six days, three grains of strychnia daily without injurious consequences—the dose having been gradually raised (Gaz. Méd. Mai, 1845); while one grain of strychnia is commonly regarded as a fatal dose to a healthy person. In a case of tetanus, Dupuytren gave as much as two ounces of opium at a dose (60 grammes), without serious consequences. (Flandin, Traité des Poisons, i, 231.) It has also been remarked, that persons affected with tetanus are not easily salivated by mercury. (Colles's Lectures, i, 77.) The effect of certain diseases of the nervous system as well as of habit, either in retarding the appearance of symptoms, or by *tolerance* in blunting the operation of a poison, it is not difficult to appreciate; and they are cases which can present no practical difficulty to a medical jurist. (See Influence of Tolerance, ante, p. 93, 100.)

On the other hand, in certain diseased states of the system, there is an increased susceptibility to the action of poison, or what is termed *intolerance* of certain drugs. Ordinary medicinal doses may in such cases exert a poisonous action. Thus, in persons who have a tendency to apoplexy, a small dose of opium may act more quickly and prove fatal. In one laboring under inflammation of the stomach or bowels, there would be an increased susceptibility of the action of arsenic or other irritants. An instance of the influence of disease in increasing the operation of poison, is occasionally seen in cases of diseased kidney (granular degeneration), in which very small doses of mercury have been observed to produce severe salivation, leading to exhaustion and death (ante, p. 101). A knowledge of this fact is of importance in reference to charges of malapraxis, when death has arisen from ordinary doses of calomel administered to persons laboring under this disease. (A medico-legal case in which this question arose, will be found fully reported in Guy's Hospital Reports, vol. iv, Oct. 1846, p. 443.) Small doses of mercurial medicines have frequently produced fatal salivation in children recovering from measles and other eruptive diseases. As a general principle, it may be affirmed, that whenever the body is much debilitated by disease, poisons acquire greater virulence of action. These facts connected with the influence of disease are obviously of some importance in relation to those cases where the person who has taken the poison is already in a diseased or exhausted state. Thus, then, there are but few exceptions to the rule laid down, that the symptoms of poisoning are liable to appear suddenly; and that in most cases they are manifested within an hour after the substance has been taken.

Symptoms appear during a state of health.—Symptoms of poisoning often manifest themselves in a person while in a state of *perfect health*, without any apparent cause. This rule is, of course, open to numerous exceptions, because the person on whose life the attempt is made, may be actually laboring under disease; and under these circumstances, the symptoms of poisoning are so obscure as often to disarm all suspicion. When poison is administered in medicine to a person laboring under illness, a practitioner is liable to be deceived, especially if the disease under which the patient is laboring be of an acute nature, and attended by symptoms of disorder in the stomach or bowels. Several cases of poisoning have occurred within a recent period in which arsenic was criminally substituted for medicine, and given to the patients while laboring under a disorder of the bowels. Hence it may be said, with respect to this character of poisoning, that when in a previously healthy person, violent vomiting and purging occur suddenly, and without any assignable cause, such as disease or indiscretion in diet, to account for them, there is strong reason to suspect that irritant poison has been taken. When a person is already laboring under disease, we must be especially watchful on the occurrence of any sudden change in the character or violence of the symptoms, unless such change can be easily accounted for on common or well-known medical principles. In most cases of criminal poisoning, we meet with alarming symptoms without any obvious or sufficient natural cause to explain them. The practitioner is of course aware that there are certain diseases which are liable to occur suddenly in healthy people, the exact cause of which may not at first sight be apparent; therefore, this criterion is only one among many on which a medical opinion should be founded.

It has been said that the symptoms of poisoning are characterized either by a *regularity* of increase, or by their becoming more and more aggravated as the case advances; but this is a weak criterion. In the operation of most of the active irritants, there are often remissions, and occasionally intermissions of the symptoms, so as to give rise to a false hope of recovery. It must not, therefore, be inferred that a recurrence of the symptoms of irritation is necessarily indicative of the administration of a fresh dose of poison. The character of the symptoms is in some cases liable to be suddenly changed; vomiting may cease, and may be succeeded by coma. While, then, on the one hand, such a case might, by our trusting too much to this criterion, be regarded as one rather of disease than of poisoning; there are, on the other hand, certain diseases which are very rapid and violent in their progress,—and the symptoms of these might, for the same reason, be mistaken for those of poisoning.

The observations here made chiefly refer to irritant poisoning; but they apply with equal force to the administration of neurotic poisons.

If a person in health is suddenly seized with stupor, convulsions, delirium, or insensibility, we have just ground for suspicion, unless some natural cause be apparent. Many forms of nervous disease may attack a person in health suddenly, and therefore a careful observation of the symptoms should be made in reference to their mode of commencement, nature, progress, duration, amenability to treatment, and result.

2. IN POISONING, THE SYMPTOMS APPEAR SOON AFTER A MEAL, OR SOON AFTER SOME SOLID OR LIQUID HAS BEEN TAKEN.—This is by far the most important character of poisoning in the living body. It has been already stated, that most poisons begin to operate within about an hour after they have been swallowed; and although there are a few exceptions to this remark, yet they occur under circumstances easily to be appreciated by a practitioner. Thus, then, it follows, that, supposing the symptoms under which a person is laboring, to depend on poison, the substance has most probably been swallowed either in food or medicine, from half an hour to an hour previously. It must be observed, however, that cases of poisoning may occur without the poison being introduced by the *mouth*. Oil of vitriol has been thrown up the rectum in the form of injection, and has caused death; the external application of arsenic, corrosive sublimate, and cantharides has destroyed life. In one case, arsenic was introduced into the vagina of a female, and she died in five days under all the symptoms of arsenical poisoning. (Schneider, Ann. der Ges. Staatsarzneikunde, i, 229.) In another instance (Reg. v. Wooler) there was reason to believe that arsenic was administered to the deceased in an enema. I found arsenic in a portion of wadding taken from the piston of an instrument used for the injection. Such cases are rare, but nevertheless the certainty that they have occurred where their occurrence could hardly have been anticipated, shows that in a suspicious case, a practitioner should not deny the fact of poisoning, merely because it is proved that the patient could not have taken the poison in the usual way,—by the mouth.

Again, persons may be destroyed by the vapors of ether, chloroform, amylene, prussic acid, or other powerful volatile poisons, introduced into the system through the lungs. Such a mode of suicide, or murder, might disarm suspicion, from the fact of no noxious material being found in the stomach. An act of Parliament has been passed, which makes it felony to administer, or even to attempt to administer, poisons in this manner (14 and 15 Vict. c, 19, sec. 111).

Let us suppose, however, the circumstances to have been such that these secret means of destruction could not have been resorted to, and that the poison is one of those most commonly selected by a murderer, such as arsenic, oxalic acid, or corrosive sublimate; then we may expect that this character of poisoning will be made evident to us, and that something must have been swallowed by the patient shortly before these alarming symptoms appeared. By observations attentively made, it may be in our power to connect the appearance of the symptoms with a particular article of food, and thus indirectly lead to the detection of a criminal. Supposing that many hours have passed since any solid or liquid was taken by a person, without any effect ensuing,—it is probable that the symptoms are due to some other cause, and not to poison. The *time of the occurrence of symptoms* in relation to a particular meal, is then a fact of especial importance in forming an opinion when poisoning is suspected,—as the following cases will show.

Cases.—The Crown Prince of Sweden was considered by many to have been killed by poison. The prince, it appears, was reviewing some troops at Stockholm, in May, 1810, when he was observed to fall suddenly from his horse, and he died in half an hour afterwards. His physician, Dr. Rossi, was accused of having administered poison to him, and he was obliged for his own security to quit the country. It is obvious, however, from an examination of the particulars of the case, that had this sudden attack been due to poison, it could have been only one of the most active narcotics, given to him but a short time before he fell from his horse. But it was ascertained that the prince had taken neither solid nor liquid of any kind for at least *four hours* previously to his death. The allegation of poisoning was thus disproved, for no poison, operating with symptoms like those under which the prince had died, could have had its effects suspended for four hours. The cause of death was apoplexy.

A child between two and three years of age, in tolerable health, was one afternoon suddenly seized with stupor, convulsions, and insensibility, and died in twenty-three hours. After death the brain was found highly congested. The suspicion of narcotic poisoning was done away with by the fact that the child had taken nothing since its dinner at two o'clock, and the symptoms suddenly appeared at half-past five, *i. e.*, three hours and a half afterwards. (Med. Gaz. xxxvi, 32.)

If in either of these cases the symptoms had supervened shortly after food had been taken, it is easy to understand that they might have been referred to poison. By bearing in mind the period at which the more common poisons begin to produce their effects, it may often be in our power to determine summarily, without a chemical analysis, whether the case be one of poisoning or not. In several instances, which have been brought to Guy's Hospital, where narcotic poison was suspected to have been the cause of comatose symptoms and rapid death, there was no difficulty in deciding against the suspicion of poisoning, merely from observing the circumstances under which the attack took place.

The subjoined case was communicated by Mr. George, of Bath, to the Provincial Journal (January 24th, 1849):—A girl, æt. 16, who was pregnant, complained of a painful swelling of the leg. On the day of her death she made a hearty dinner of beef, vegetables, and porter, with the family at one o'clock, and remained in the same room where she had partaken of that meal until three o'clock. On quitting the apartment, she began to groan, complained of pain at the pit of the stomach, and became faint: she vomited, and in three-quarters of an hour she died. On inspection, there was an inflamed appearance both of the duodenum and stomach. A careful analysis of the viscera as well as of the matter vomited revealed no poison; and the fact that no symptoms had occurred during a period of *two hours after the meal*, strongly corroborated the conclusion that deceased had died from natural causes.

Facts of this kind may sometimes serve to establish the innocence of an accused party, and at others to point out the real criminal. A woman aged sixty-five, accused her husband, an old man of seventy, of having attempted to poison her. The woman was passionate, ill-tempered, eccentric in her habits, and subject to occasional attacks of hysteria. She handed to the authorities a vessel containing arsenic in coarse powder; and some food which she stated had been prepared for her by the prisoner. On analysis, the food was found to contain a large quantity of arsenic. The husband was immediately committed to prison. The wife left her bed, and was apparently quite well; and so she remained for eight days afterwards, no symptoms of poisoning having manifested themselves. She was then seized with a fit of mania, and was guilty of many extravagant acts. She died the following day, *i. e.* nine days after she had accused her husband of having administered arsenic to her in her food. On an examination of the body, it was evident she had died from the effects of arsenic. This poison was found in large quantity in the alimentary canal; and there were the usual morbid changes in the stomach and intestines. The husband denied that he had administered poison to the deceased. This denial, however, would have availed him but little, had it not been for the careful medico-legal investigation of the whole case, made by the medical witnesses. As the husband had been confined in prison *eight days* before the death of his wife, he could not have committed the crime imputed to him, unless he had administered the arsenic previous to his imprisonment. His guilt, therefore, rested upon the medical question, whether a large quantity of arsenic could be taken by a person and remain dormant in the system, without producing any of its usual effects for the long period of eight days? The witnesses very properly answered the question in the negative, and the husband was immediately discharged. (Annales d'Hygiène, 1836, ii, 391.) While the prisoner was with his wife, she did not suffer from the symptoms of poisoning, nor was there any proof that he had administered poison. When, however, he was so situated that he could not possibly have been accessory to its administration, she died from its effects. It was fortunate for the accused that he was thrown into prison, and that the case fell into the hands of persons versed in the subject of legal medicine.

Jean Aitkin, or Humphreys, was tried at the Aberdeen September Circuit, 1830, for the murder of her husband, by pouring sulphuric acid down his throat as he lay asleep in bed. The parties frequently quarrelled, and were both addicted to habits of intoxication. On the night in question, some friends had passed the evening with them, drinking. They left the house about twelve o'clock at night, and soon after this, the deceased was seen asleep in bed. The only persons in the house at this time were the prisoner and a servant-maid, and the street door was locked, so that no other person could have had access. The prisoner left the servant's room on her stocking-soles, a thing unusual for her, and when she returned in about twenty minutes, she told the servant that her husband was roaring mad with drink. The girl, upon going to him, found him lying upon his back, declaring that he was all roasting. The prisoner at first showed an unwillingness to send for a medical man, but at length did so. When the deceased left his guests at twelve, there were only two glasses on the table in the room; but when the neighbors came in after the alarm, there were *three*, and the third was proved to have come from a room above stairs, of which the prisoner had the key. This glass contained, it was supposed, sulphuric acid. In the room where the deceased was lying there was a phial which had contained sulphuric acid, but it was then nearly empty. The deceased lived two days, but never could give any further account of the matter than that he went to sleep quite well, and awoke "all roasting," and had suffered the utmost agony ever since. He evidently died from the effects of sulphuric acid, large quantities of which were detected on his shirt, on the blanket and bedcover, and a little on the prisoner's bedgown and handkerchief; but not a trace of the poison could be discovered in the stomach or intestines of the deceased. (Alison, Criminal Law of Scotland, p. 75; also Medical Gazette, vol. viii, p. 77.) In the defence, it was alleged that the deceased had voluntarily taken the poison and committed suicide; but the only time at which he could by any possibility have taken it, was when he was drinking with his friends; for immediately after they left, he went to bed, and was seen asleep; and, according to his own account he awoke suddenly with the pain and other symptoms produced by this poison. It was impossible that he could have swallowed the acid while drinking with his friends; for the symptoms of the corrosives come on *suddenly*, and cannot be suspended; therefore, the poison must have been poured down his throat while he was sleeping, and as the house was at that time fastened up, this act could only have been perpetrated by the prisoner or the maid-servant. The circumstances above mentioned clearly showed that the prisoner was the guilty party. It will be observed that all suspicion of suicide, as well as of murder on the part of the persons with whom the deceased had been drinking, was entirely removed, by attention being paid to this well-marked character of the corrosive poisons.

Valuable as this character is to a medical jurist in leading him to a correct opinion in a case of suspected poisoning, if overstrained it may be productive of much mischief. In the case of Reg. v. Palmer (Central Criminal Court, May, 1856), it was contended, on the part of the prosecution, that the deceased Cook had died from a dose of strychnia administered in a pill by the prisoner. For the defence, two medical witnesses, Dr. Letheby and Mr. Nunneley, were called, and they deposed, that in their opinion the deceased had not died from strychnia, because, *inter alia*, the length of time which had elapsed before the symptoms came on, after the deceased had swallowed the alleged poisoned pill, was greater than is observed in cases of poisoning by strychnia. The interval was proved to have been an hour, or not more than an hour and a quarter, the deceased having fallen asleep (see ante, p. 108, on the effects of sleep), and having suddenly awoke from the access of the symptoms. The medical facts were very simple. Cases of fatal poisoning by strychnia have been so few, that it is not possible to fix with accuracy even an average time for their appearance. The interval must vary, as with other poisons, according to dose, and many circumstances. One case of poisoning by strychnia was cited on the trial, in the hearing of these witnesses, in which the symptoms did not appear until *one hour* after the poison was taken; another case is recorded (Ed. Monthly Journal, Feb. 1848, p. 566), in which no symptoms appeared for two hours and a half! In a third case, that of Assistant-Surgeon Bond, recently communicated to me from India (May, 1858), the deceased took by mistake two pills containing two grains of strychnia, and was two hours in bed before he suddenly awoke with the tetanic symptoms indicative of poisoning by strychnia. The opinion thus loosely given by these witnesses was based on experiments on animals! It was not only incorrect, but, under the circumstances, unjustifiable, as the case of one hour's interval in the human subject, quoted from the Lancet at the trial, was safer for guidance on such a question than the conflicting results of a hundred experiments on dogs and rabbits. The admission of this scientific truth would, however, have seriously damaged the case set up for the prisoner.

When symptoms, resembling those of poisoning, speedily follow the introduction of food or medicine into the stomach, there is always great room for suspicion; but caution should be observed in drawing inferences, since extraordinary coincidences sometimes present themselves. In the case of Sir Theodosius Boughton, who was poisoned by his brother-in-law, Donellan, in 1781, the fact of alarming symptoms coming on in *two minutes* after the deceased had swallowed what was supposed to be a simple medicinal draught, became a most important piece of evidence. There is no doubt that laurel-water had been substituted for the medicine by the prisoner. (See post, PRUSSIC ACID.) The practice of substituting poisonous mixtures for medicinal draughts, or powders, is by no means unusual, although it might be supposed to indicate a degree of refinement and knowledge not commonly to be found in the lower class of criminals. In other cases, poison may have been ignorantly dispensed for medicine. Medical practitioners may thus be fatally deceived. The late Baron Alderson, on one occasion, publicly related the following case: An apothecary prepared a draught, into which another person put poison, intending thereby to destroy the life of a patient for whom the medicine was prescribed. The patient, not liking the taste of the draught, and thinking that there was something suspicious about it, sent it back to the apothecary, who, knowing the ingredients of which he had composed it, and wishing to prove that he had done nothing wrong, drank it himself, and died. In this case, he was the unconscious agent of his own death; and, although the draught was intended for another, the person who poisoned it was held guilty of murder.

The following case illustrates the fatal effects arising from a mistake made unconsciously: A druggist, in Baltimore, U. S., prepared some medicine, for a child, from a prescription. The child took a dose, and died instantly. The druggist, on being questioned, declared that he had made no mistake, and was so confident in his accuracy that he swallowed a portion of the medicine, and in five minutes afterwards he was dead! On analysis, it was found that he had, by some mistake, put into the mixture sufficient prussic acid to kill fifty persons! (Lancet, February 14, 1857, p. 182.)

We should, therefore, remember that, on these occasions, poison may have been ignorantly or criminally substituted for an innocent medicine prescribed. In 1856, a physician, of this metropolis, nearly lost his life by drinking what he supposed to be an infusion of ash-leaves. He had prescribed this infusion for a patient who had been rendered insensible by the first dose; and, in order to satisfy himself of its nature, he drank only a small quantity. It turned out that an ignorant herb-dealer had sold the leaves of belladonna for those of the ash, and it was an infusion of belladonna which had thus been taken by the patient and physician. In the well-known case of Palmer, pills containing strychnia were substituted for pills of morphia, prescribed and compounded by the medical attendant. A medical man is not required to make himself the subject of experiment, for the purposes of justice. As it is impossible for him to say what changes may have been made in a medicine, or in the dispensing of it, he must always incur a risk by such a proceeding. A chemical analysis of the medicine, or the administration of some of it to an animal, will suffice to show, whether it does or does not contain any substance of a noxious or poisonous kind.

In a case which I was required to examine, in February, 1847, a mixture of soap-liniment and opium had been substituted for the tincture of sesquichloride of iron, prescribed for a female many months before. Some of the liniment had been given to an infant, and, as it was alleged, had led to its death. The mother was tried and acquitted upon a charge of murder at the Essex Lent Assizes, 1847, the cause of death not being clearly traced to the action of the opiate. (Queen v. Gray and Bright.) The defence was, that a mistake had been made in dispensing the medicine; but the fact that the phial had originally contained an iron-mixture, was proved by the discovery of iron in some brown stains upon the label, owing to a portion of the liquid having been accidentally spilled over it. The cork was also blackened by the gallic acid and tannin contained in it, from contact with the original iron-mixture.

The occurrence of symptoms resembling those produced by poison, soon after a solid or liquid has been taken, may be a pure coincidence. In such a case, poisoning is always suspected by the vulgar; and it will be the duty of a medical jurist to guard against the encouragement of such a suspicion, until he has strong grounds for believing it to be well founded. No public retraction or apology can ever make amends for the injury which may in this way be inflicted on the reputation of another; for those who hear the accusation may never hear the defence. In such cases, a practitioner may entertain a suspicion, but he should always avoid *expressing* it,—giving it publicity, or encouraging the expression of it by others. When death is not a consequence, it is difficult to clear up such cases, except by the aid of a chemical analysis; but this, as we know, is not always applicable. If death ensue, the real cause is usually apparent, and a suspicion of poisoning may be thus removed by an examination of the body and an analysis.

But the poisonous substance may be found in the body and yet the death have been a pure coincidence. In a case in which I was consulted, three grains of arsenic were given by mistake for calomel to a child, while in a dying state. The child died soon afterwards; the discovery of arsenic in the stomach led an analytical chemist to assert that the child had died from this poison, and two females, by reason of this chemical opinion, were tried upon a charge of murder, but acquitted, without being called upon for a defence. (See the case of Dore and Spry, post.) The chemist adopted the theory that calomel was really given; but as none was found by him in the stomach, and it must have been found, if it had been given, it was clear that he had made a mistake.

Certain symptoms may follow the use of food containing no poison, and yet, from taste or smell, as well as the coincidental effects, there will be a strong disposition to charge the crime of poisoning on others. Many cases of this kind have presented themselves to me, in which the idea of poison had been taken up and so persisted in, that even the results of a chemical investigation were doubted. In such cases the symptoms appear to result from the force of imagination. A man suffered from shivering and nausea, with a strong disposition to vomit, after drinking some cider. He assigned these symptoms to poison in the cider. A formal investigation was made by M. Chevallier and others, which resulted in the discovery that some common salt had been mixed with the cider, and had given to it a peculiar taste! (Annales d'Hygiène, 1854, ii, p. 423.)

In the case of a Mr. Brettle, who met with an accident in Paris, in 1856, which led to his death from effusion of blood on the brain, an English physician, who was in attendance, was charged with having accelerated his death by poison. It appears that while the deceased was in a hopeless condition, this physician took from his pocket and administered to him a white powder (three-quarters of a grain of tartarized antimony), and the deceased died two hours afterwards. There were no symptoms produced by the powder,—the deceased gradually sank from the effects of the accident,—and the French physicians who examined the body, reported that death had been caused by the accident. In short, there was no ground for imputing death to poison, either directly, or indirectly, by accelerating the impending fatal results of the accident. Such a charge should never be made, except upon the most conclusive evidence.

The fatal symptoms produced by perforation of the stomach, which in some respects resemble those of arsenical poisoning, almost always attack an individual soon after a meal. When they occur some hours afterwards, there is less likelihood of confounding them with arsenic. A few years ago, Mr. Hilton and myself were required to examine judicially a case of this description. Our judgment was in a great measure aided by the fact, that the violent symptoms did not appear until about three hours after a meal. An instance occurred within my knowledge, where an aged lady took three grains of a white powder, prescribed for her by her medical attendant. In about ten minutes afterwards she was seized with coma, and died in the course of an hour. The medicine which she took was sulphate of quinine. In such a case it might have been most plausibly said,—morphia or some other poisonous alkaloid had been swallowed; but the circumstances were well known; death was due to apoplexy. In another instance, a woman, aged 37, rose in the morning in her usual health, with the exception of having a slight headache; immediately after taking breakfast she was attacked with violent vomiting, which continued for half an hour,—she then fell down and died suddenly.

Here again there was room for suspecting poison, owing to the time of the occurrence of symptoms, but it was proved that the woman had died of disease of the brain.

It is not improbable that the mere fact of a person eating a full meal after *long fasting* may give rise to symptoms resembling those of irritant poisoning. Mr. Holland, of Manchester, has communicated to me two cases of this description, in one, the symptoms were very violent, and the patient nearly died,—in fact was laid out for dead. The other patient suffered from severe pain in the stomach for several weeks. Poisoning was at first strongly suspected, but the suspicion was removed by the fact, that others in health had partaken of the same food, principally potatoes mixed with gravy, without any injury; and there was no reason to suppose that any irritant poison could have been mixed with the food. The two who suffered were extremely weak and exhausted from long fasting, and were observed to eat their food which was quite wholesome, voraciously. The effects of *cold liquids* in producing sudden death resembling the action of poison, have been elsewhere referred to (ante, p. 25). The fatal effect is here due to a shock produced on the nervous system.

3. IN POISONING WHEN SEVERAL PARTAKE AT THE SAME TIME OF THE SAME FOOD OR MEDICINE (MIXED WITH POISON) ALL SUFFER FROM SIMILAR SYMPTOMS.—This character of poisoning cannot always be procured; but it furnishes good evidence of the fact when it exists. Thus, supposing after a meal made by several persons from the same dish, only one suffers, the suspicion of poisoning is considerably weakened. The poisoned article of food may be detected by observing whether they who suffer under symptoms of poisoning, have partaken of one particular solid or liquid in common. In a case of accidental poisoning at a dinner-party, it was observed that the persons who suffered from the symptoms, had taken port wine only: the contents of the bottle were brought to me for examination; they were found to consist of a saturated solution of arsenic in wine. In general, considerable reliance may be placed upon this character, because it is improbable that any common cause of disease should suddenly attack with violent and alarming symptoms, several healthy persons at the same time, and within a short period after having partaken of food together. We must beware of supposing that when poison is really present, all will be attacked with precisely similar symptoms, or at the same interval of time; because, as we have seen, there are many causes which may modify these conditions. In general, that person who has partaken most freely of the poisoned dish will suffer most severely, but even this does not always follow. There is a well-known case recorded by Bonnet, where, among several persons who partook of a dish poisoned with arsenic, they who had eaten little and *did not vomit*, speedily died; while those, on the other hand, who had partaken largely of the dish, and had in consequence vomited freely, recovered. In the alleged poisonings by arsenic at Hong-Kong (1857), the large quantity of poison introduced into the food supplied to the family of Sir John Bowring and others, appears to have acted in a similar manner.

It was just now remarked, that there is no disease likely to attack several healthy persons at the, same time, and in the same manner. This is undoubtedly true, *as a general principle*, but the following case will show that mistakes may occasionally arise even under these circumstances. It occurred in London, during the prevalence of the malignant cholera in the year 1832. Four of the members of a family living in a state of great domestic unhappiness, sat down to dinner in apparently good health; some time after the meal, the father, mother, and daughter, were suddenly seized with violent vomiting and purging. The stools were tinged with blood, while the blueness of the skin, observed in cases of malignant cholera, was wanting. Two of the parties died. The son, who was known to have borne ill-will against his father and mother, and who suffered no symptoms on this occasion, was accused of having poisoned them. A strict investigation took place before the coroner; but it was clearly shown by the medical attendant, that the deceased persons had really died of the malignant cholera, and there was no reason whatever to suspect that any poison had been administered to them. In this instance, it will be perceived that symptoms resembling those of irritant poison appeared suddenly in several persons in perfect health, and shortly after a meal. We hereby learn that the utility of any rules for investigating cases of poisoning, depends entirely on the judgment and discretion with which they are applied to particular cases.

Instances in which a whole family are simultaneously attacked with symptoms resembling those of poisoning, occasionally present themselves, and are often attended with great obscurity. The case of the Arzone family is, in this respect, worthy of notice. The family, consisting of the father, mother, and three children, were in good health up to the evening of December 30, 1840. On January 1, 1841, the father, a manufacturer of colors, was suddenly taken ill with griping pains and purging, which never ceased until death. He was sick at times, but never vomited; the motions were offensive and black; he had frequently cold fits during the day, followed by much fever. His joints were swollen and painful. He died on the 20th January. On inspection, the stomach and intestines were found healthy. The lungs and pleura presented strong evidence of inflammation sufficient to account for death. The three children, as well as the mother, after suffering from somewhat similar symptoms, died,—the death of the mother being accelerated by parturition. All complained of general soreness of the fleshy parts of the joints, great sensibility of the skin, and pain produced by the least change of posture; they suffered from diarrhœa, complained of a cankery or metallic taste in the mouth, and there was a watery state of the mouth and eyes, with a dry cough. The abdominal viscera showed no change from the normal state, but the lungs were congested. (Med. Gaz. xxx, 326.) The symptoms could not be referred to any epidemic or malaria; the man, to my knowledge, lived in a healthy situation, and none of his neighbors were attacked. It was therefore difficult to ascribe them to malaria, or any disease depending on natural causes, occurring simultaneously in so many members of a family, previously healthy. On the other hand, there was no evidence that poison had been taken in the food, and the appearances in the body were not indicative of the action of any irritant, yet the symptoms and appearances were in some respects compatible with the hypothesis of chronic poisoning, either from the slow inhalation of the fine powder used in the preparation of colors, or from their introduction into food through want of cleanliness. There was no doubt that he employed the arsenical cobalt ore (the Tunaberg ore, see post, ARSENIC) in the preparation of a kind of ultramarine; and possibly, as it was suggested at the time, impalpable dust in the preparation of this substance may have given rise to the symptoms. The question of poisoning could have been fairly solved in this case only by a minute analysis of the tissues; but the processes for detecting mineral poisons in the tissues were then not known. We shall see hereafter (ARSENITE OF COPPER, CARBONATE OF LEAD) that these poisonous pigments may penetrate into the system in an insidious manner, and produce serious effects. Walls painted, or even papered with arsenical pigments, have thus caused symptoms of poisoning in those who inhabited the apartments. It is not at all improbable that the oil of turpentine, which is largely used as a medium for colors, may by its volatility become a vehicle for the diffusion of a poisonous pigment.

The simultaneous occurrence of symptoms terminating fatally in two or more persons, is always well calculated to excite grave suspicions of poisoning; and a safe opinion can then only be formed by noting the character of the symptoms, or, if this source of evidence be wanting, by the detection of poison in the food or bodies of the individuals. A simultaneous attack merely furnishes a presumption in favor of poisoning, to be supported or rebutted by other circumstances. A case, which will be more particularly described hereafter (see CONVULSIONS), was referred to me in December, 1846, by Mr. Wood, coroner for Surrey, in which two children, previously healthy, died under similar symptoms, very suddenly, and after a short illness. It was reasonably suspected, in the first instance, that narcotic poison had been given to them; but an examination of the facts of the case, as well as an analysis of the food and contents of the stomachs, proved that poison was not the cause, and thus removed a heavy load of suspicion from the parents.

Obscure symptoms of poisoning may occur simultaneously in several members of a family from accidental causes, the nature of which may not be even suspected. Thus, various articles of food may be poisoned by copper through want of cleanliness in the use of culinary utensils (see COPPER); or the water supplied to a house may be contaminated with lead from the use of that metal in pipes, cisterns, or merely as a cover to a tank (see CARBONATE OF LEAD). The safety of the individuals, and probably the exculpation of an innocent person, wrongly accused of poisoning, will depend on the acumen of the medical attendant in discovering the real cause. In reference to this question, the case of *Solomon* v. *Lawson*, tried at the Surrey Lent Assizes, 1845, is of some medico-legal interest. It was alleged that the defendant had libellously stated that the plaintiff, who was in the habit of supplying water to ships at St. Helena, had supplied some water poisoned with lead or copper to a particular ship, and that this was the cause of an illness which had prevailed among the passengers on the homeward voyage. It appeared in evidence that five of the officers and the steward were affected, and two of the officers suffered severely from symptoms which, although slightly varying in the respective cases, the surgeon of the ship did not hesitate to refer to some metallic poison. Two medical men who attended three of the passengers on their return to England, stated that in their opinion the effects were due to lead, which was rendered probable by the fact that partial paralysis of the lower extremities and blueness of the gums were among the symptoms. Some of the crew, however, did not suffer, although stated to have used the same water. The water, as it was supposed but not proved, was analyzed, and no lead or copper was found in it. As so much depended on this analysis, it is to be regretted that the identity of the sample was not more clearly made out, and that it was not assigned to some experienced chemists. It is impossible, I think, to draw any other conclusion from the evidence, than that drawn by the medical witnesses, *i. e.* that the cause of the symptoms was really owing to some metallic poison, probably to lead. The jury, however, held, under the direction of the judge, that the water was not *proved* to have been poisoned, and returned a verdict, with very heavy damages, against the defendant. They were probably misled by the piece of evidence regarding the immunity of some who had used the water. It is a mistake to suppose that in this insidious form of poisoning, either all must suffer from the effects, or there is no poisoning at all! Persons exposed to the same influence of chronic poisoning by lead, are very differently affected. In the case of the royal family of France, at Claremont, in 1849, although the whole household was supplied with the same water, only thirteen out of thirty-eight members of the family suffered from the effects of lead-poison. The cause was in this case, medically speaking, very clear, but the water might have been legally pronounced free from poison, because twenty-five persons had escaped!

It may be here proper to remark, that the water of wells in the neighborhood of chemical works is often impregnated with poison. Persons who unsuspectingly use this water may be attacked with symptoms of poisoning, and die from the effects. In the Registrar-General's Quarterly Report for 1846, it is stated that nearly the whole of the members of a family in Derbyshire, died from having drunk water impregnated with arsenic which was drawn from a well contiguous to certain chemical works attached to the premises. (Med. Gaz. xxxvii, 843.)

It is proper to bear in mind, in conducting these inquiries, that symptoms resembling those produced by irritant poison may be occasionally due to the food which may have been taken by a family at a meal. Besides flesh rendered unwholesome from disease and decay, there are certain kinds of shell-fish, as well as pork, bacon, sausages, cheese, and bread, which, under certain circumstances, may give rise to serious symptoms, and even death. In such a case, all the foregoing characters of poisoning are brought out; and, indeed, it may be regarded as one of poisoning by an animal or vegetable irritant. These cases present some difficulties; great ambiguity frequently arises, from the fact that not more than one or two persons may be affected, who have frequently before partaken of the same kind of food without any particular inconvenience (see ANIMAL IRRITANTS).

4. THE DISCOVERY OF POISON IN THE FOOD TAKEN, OR IN THE MATTERS VOMITED.—One of the best proofs of poisoning in the living subject is the detection of poison by chemical analysis, either in the food taken by the person laboring under its effects, in the matters vomited, or in the urine, if the poison be one of those which are eliminated by the kidneys. The evidence is, of course, more satisfactory when the substance is discovered in the matters vomited, than in the food; because this will show that poison has really been taken, and will at once account for the symptoms. If these sources of evidence are not accessible, then we must examine the food of which the patient may have partaken. Should the results in all cases be negative, it is probable that the symptoms may have been due to disease. In investigating these cases in the living subject, a medical jurist must remember that poisoning is sometimes *feigned*, and at others, *imputed*. It is very easy for an artful person to put poison into food, and to accuse another of having administered it, as well as to introduce it into the matters vomited or discharged from the bowels, or into the urine. There are few of these accusers who go so far as to swallow poison under such circumstances, because there is in general a great dread of poisonous substances; and it will be at once apparent, that it would require a person well versed in toxicology, to feign a series of symptoms which would impose upon a practitioner at all acquainted with the subject. In short, the difficulty reduces itself to this: What inference can we draw from the mere chemical detection of poison in food? A medical man may say whether poison is or is not present in a particular article of food; but he must leave it to the authorities of the law, to develop the alleged attempt at administration. If the poison should have been actually administered, then we may expect to find the usual symptoms. With regard to the detection of poison in the matters vomited from the stomach, this affords no decisive proof that it has been swallowed, except under two circumstances: 1. When the accuser actually labors under the usual symptoms of poisoning, in which case there can be no feigning, and the question of imputation is a matter to be established by general evidence. 2. When the matters are actually vomited into a *clean vessel* in the presence of the medical attendant himself, or of some person on whose testimony perfect reliance can be placed. (For a case of imputed poisoning in which the accused party had a narrow escape of his life, see ante, p. 113.)

CHAPTER IX.

EVIDENCE FROM THE NATURE OF THE SYMPTOMS—DISEASES RESEMBLING IRRITANT POISONING—CHOLERA—GASTRITIS—ENTERITIS—GASTRO-ENTERITIS—PERITONITIS—ULCERATION AND PERFORATION OF THE STOMACH—STRANGULATED HERNIA—INTUSSUSCEPTION—INTERNAL STRANGULATION OF THE INTESTINES—DISEASES RESEMBLING NARCOTIC POISONING—APOPLEXY—SUDDEN DEATH FROM NATURAL CAUSES—EPILEPSY—ATELECTASIS—TETANUS—CONVULSIONS IN INFANTS—DISEASES OF THE BRAIN AND SPINAL MARROW—OF THE HEART—DEATH FROM DISTENSION OF THE STOMACH—RUPTURES OF THE GALL-BLADDER.

Nature of the symptoms.—The nature and order of occurrence of the symptoms under which a person is laboring should be accurately observed in a suspected case. In poisoning, the symptoms are commonly well marked, and have a peculiar character; those of disease are less certain, and are more likely to create embarrassment. Owing to this, it happens that in practice, disease is much more liable to be mistaken for poisoning, than poisoning for disease. An account of the symptoms produced by the two classes of poisons will be found at page 104; and the special details,—in the description of each poison respectively. At present it will, therefore, only be necessary to enumerate, on the one hand, those diseases, the symptoms of which might be mistaken for irritant poisoning; and, on the other, those which might be mistaken for narcotic poisoning.

DISEASES RESEMBLING IRRITANT POISONING.

The diseases, the symptoms of which resemble those produced by *irritant* poisons, are cholera, gastritis, enteritis, gastro-enteritis, peritonitis, perforation of the stomach or intestines, strangulated hernia, colic, and hæmatemesis.

Cholera.—This name is given to a disease in which there is a combination of vomiting and purging, generally of biliary matter. It is necessary to distinguish the common English cholera from the Asiatic or malignant form of the disease. In the Asiatic cholera there is usually sudden and extreme prostration of strength; the surface of the body is cold, and it sometimes has a dark livid or leaden hue, especially observed in the skin of the hands and feet. The skin is shrivelled, the features are pinched, the breath is cold as it issues from the mouth; the matters discharged from the bowels are very copious, resembling rice water with flakes of coagulated mucus floating in them. There is the most intense thirst, and the patient will drink a large quantity of cold water. The symptoms of poisoning by arsenic and other irritants are wholly different from these, if we except perhaps the intense thirst, which is present in both cases. Dr. Wilks met with one case of poisoning by arsenic, which proved fatal in nine hours, in which the symptoms were similar to those of malignant cholera. (Guy's Hosp. Rep. 1855, p. 364.)

In poisoning by arsenic the skin is hot and cold at intervals; the pulse frequent, small, and irregular, amounting to from 120 to 130 in a minute. It is only in the last stage of arsenical poisoning (collapse) that there is an icy coldness of the limbs. With the thirst there is commonly great constriction in the throat, not met with in this form of cholera.

The common English cholera, as it occurs in summer and autumn, more closely resembles irritant (arsenical) poisoning in its symptoms. Thus an attack often comes on in a healthy person in about half an hour after a meal. It is accompanied by vomiting and purging of bilious liquid, and by violent pain in the abdomen, continuing until death when the case terminates fatally. It may usually be traced to some indigestible food of which the patient has partaken. Many acquittals on criminal charges have taken place from the great difficulty which exists in distinguishing this last-mentioned form of cholera from arsenical poisoning; and, in truth, it may be observed, that if in any case medical evidence rested on symptoms alone, it would be scarcely possible, in some instances, to draw such a clear distinction between the symptoms of this disease and those of poisoning, as the law would deem absolutely necessary for conviction on a criminal charge. The rules recommended for forming an opinion, as they are laid down by the best writers on toxicology, are not satisfactory. Perhaps the following may be taken as a statement of the most striking differences. In irritant poisoning the evacuations are often tinged with blood; in cholera they are not tinged with blood, but commonly deeply colored by bile. In irritant poisoning, these evacuated liquids will sooner or later yield traces of poison when analyzed. In cholera this is of course not the case. The attack of cholera is commonly dependent on some irregularity of diet, and appears chiefly in summer and autumn. Irritant poisoning may occur at any season. Except when it prevails in a severely epidemic form, from intense heat or other causes, and attacks the very aged or the very young, English cholera is not often fatal; and when it does prove fatal, it is commonly after three or four days from its commencement, by exhaustion of the patient. In irritant (arsenical) poisoning, death is a common result in twenty-four hours, when the symptoms produced by the poison are such as to resemble those of cholera, *i. e.* poisoning in its most acute form. In irritant poisoning, the symptoms usually come on in about half an hour or an hour after a meal; and although cholera may commence its attack at about the same period, yet, supposing several persons to have partaken of the food, all will suffer more or less if it be really a case of poisoning,—not if it be a case of cholera. It would be at least something very unusual, that several healthy persons should be attacked by cholera at the same time, unless the attack were owing to some improper kind of food used at the meal. (See case, p. 119.) Lastly, an analysis of the food may serve to determine whether irritant poison was or was not the cause of the symptoms. Of all irritant poisons, arsenic comes the nearest to cholera in the character of the symptoms. It is right to bear in mind, however, that a case of arsenical poison is often accompanied by special symptoms which are met with neither in cholera nor in any disease resembling it. Thus in persons who have taken arsenic and survived the first effects of the poison,—the conjuctivæ (whites) of the eyes often become inflamed, sometimes at a very early period,—there is also great irritation of the skin, followed by a peculiar (eczematous) eruption;—and occasionally numbness, or tingling in the hands and feet, as well as paralysis and coma, appear among the symptoms. In cholera, nothing of this kind is witnessed: hence we have in these peculiar symptoms of arsenical poisoning means for assisting us in forming an opinion. When the person dies, an examination of the body with an analysis of the contents of the stomach, or if death speedily follow the attack, an analysis of the tissues of the soft organs, will often remove any doubts that may have existed on the real nature of the case. In numerous cases, arsenical poisoning has been mistaken for cholera, and the fact of poisoning has remained concealed until an analysis was made. (See cases of Reg. v. Chesham, Essex Lent Assizes, 1847; and Reg. v. Foster, Suffolk Lent Assizes, 1847.) M. Tardieu has fully examined the medico-legal bearings of this subject. (Ann. d'Hyg. 1854, ii, p. 162.)

During life, it may be in the power of a practitioner, to procure some portion of the matters vomited or discharged by the bowels—or, in the absence of these, the urine,—and a chemical analysis may then reveal the cause of the symptoms, and prove that they are due to poison. There is reason to believe that the case of the Duc de Praslin (Paris, 1847), was at first mistaken and treated for one of cholera by the physician who saw him. He was not treated like one who was laboring under the effects of arsenic. The deceased had secretly taken poison. The first symptoms appeared in the form of vomiting and purging on the 18th of August, and he died on the 24th. On the 20th, he complained of no pain in the abdomen, even on pressure. The peculiar symptoms observed were extreme smallness and irregularity of the pulse and coldness of the surface. On the 21st the vomiting and purging had ceased. On the 22d there was an aggravation of the symptoms, some of them strongly indicative of the effects of arsenic,—spasmodic constriction of the throat, —great pain in swallowing,—intense thirst, and a burning sensation from the mouth to the anus. The mind remained unaffected until the last. (Ann. d'Hyg. 1847, ii, 390.) An analysis of an evacuation passed on the 21st, and of urine passed on the 22d, led to the discovery of the presence of arsenic, and for the first time clearly established the nature of the case. The symptoms, however, presented several special characters which might have been fairly referred to the action of arsenic.

GASTRITIS, ENTERITIS, GASTRO-ENTERITIS, PERITONITIS.—These diseases do not commonly occur without some obvious cause; indeed, the two first, in the acute form, must be regarded as the direct results of irritant poisoning. Thus arsenic and other irritants, when they prove fatal, commonly give rise to inflammation of the stomach and bowels. In all cases, in which these diseases present themselves, the object of a practitioner is, therefore, to determine the *cause* of the inflammation, whether it be due to natural disease, or the action of an irritant poison.

The distinction will chiefly rest, 1. Upon the time of the occurrence of the symptoms after a meal; 2. The order of their occurrence; 3. The obstinate constipation of the bowels, which is observed in gastritis and enteritis, as contrasted with the violent vomiting and purging met with in irritant poisoning; 4. The presence of fever in these diseases. The history of the case so clearly explains its nature, that we seldom hear of these diseases being mistaken for irritant poisoning. The same observations apply to peritonitis, in which disease there is also constipation, and but little vomiting, with general tenderness over the whole of the abdomen. It has been doubted by some pathologists, whether the diseases above mentioned can occur spontaneously, and without any apparent cause. All agree that instances of idiopathic acute gastritis are rarely observed in persons otherwise healthy. Two cases were reported to the Medico-Chirurgical Society, by Dr. Burne (Med. Gaz. xxv, 414), and another case has been published by Dr. Berncastle (Lancet, March, 1844). The symptoms were of the usual character—constant vomiting, and no purging, and rapid sinking. After death the stomach was found in a high state of inflammation, but all the other organs were healthy. A suspicion of poisoning did not attach to the case. Acute enteritis from natural causes is much more common than acute gastritis. These diseases, in a chronic form, have a very slow course, and may be a secondary result of irritant poisoning. The symptoms are unlike those produced in the acute form of poisoning. The case of Reg. v. Hunter (Liverpool Spring Assizes, 1843), was successfully defended on the theory of gastro-enteritis from natural causes, in spite of the strongest suspicions that arsenic was the cause of death.

PERFORATION OF THE STOMACH AND INTESTINES.—The symptoms attending perforation of the stomach, in some respects resemble those of irritant poisoning. They often occur suddenly to a healthy person after a meal. This disease is almost invariably fatal, and may be immediately recognized on an examination of the body. Even in the rare cases in which it is not fatal, the means of diagnosis are not difficult. (See post.)

STRANGULATED HERNIA.—It is difficult to suppose that this disease should ever be confounded with irritant poisoning. The seat of pain, with an examination of the part, would at once show the physical cause to which the symptoms were due. (See Ann. d'Hyg. 1854, vol. ii, page 153.)

INTUSSUSCEPTION OF THE BOWELS—ILEUS, ILIAC PASSION, INTERNAL STRANGULATION.—These terms are applied to a disease in which there is violent vomiting without purging—the mechanically locked state of the bowel, preventing the passage of fæces. It differs from diarrhœa, in which there is purging without vomiting, and from cholera, in which there are both. In irritant poisoning, although occasionally there may be an absence of either vomiting or purging, it is generally observed that both of these symptoms are present, and in addition acute pain, referable chiefly to the region of the stomach. In the disease referred to, the symptoms commence suddenly in a previously healthy person, and death takes place from strangulation internally. A report of a case of this kind was read by Dr. Snow before the Medico-Chirurgical Society, in June, 1846. The patient died in four days, and, on inspection, a portion of the ileum (small intestines) was found strangulated in an aperture of the mesentery (Med. Gaz. xxxviii, 1049). Two cases of a similar kind are quoted by Dr. Snow, in his paper. In the same number of that journal, is reported a fatal case in which the cause of strangulation was produced by the weight of a supplementary spleen drawing the omentum into a cord (page 1053). Other cases, in which there were various mechanical causes of internal strangulation, are reported at pp. 1073 and 1075. The symptoms of this disease are a sudden access of severe pain, chiefly confined to one spot, not in the region of the stomach, as in irritant poisoning, but in the central or lower part of the abdomen—severe and constant vomiting, at first bilious, and afterwards of fæcal matter; but, in some instances, the vomited matter is, throughout, a yellow or green-colored liquid. There is obstinate constipation, if we except what may be discharged from the lower bowel. The detection of the disease is commonly not difficult, and a careful inspection of the body will immediately reveal the cause of death. The case, in general, terminates fatally in three or four days, as there are no means of relieving the strangulation: hence evidence from appearances is rarely deficient.

In the following case (that of a friend), in which the cause of the symptoms was very obscure, the suspicion of poisoning was, in the first instance, strong. This gentleman, while in good health, was suddenly seized, two hours and a half after his dinner (taken as usual), with the most severe pain in the pit of the stomach. This was succeeded by nausea and vomiting, which continued without intermission for some hours. Twenty-four hours after the attack he was seen by a medical man: the vomiting had ceased, but everything which he swallowed was rejected. There were frequent rigors; the pulse quick and wiry; extremities cold; features sunk; voice feeble; and a cold sweat covered the body. The region of the stomach was tender, and there was a fixed pain, increased by pressure, radiating from the cæcum along the ascending colon. There was no hernia; but the abdomen was hard, and drawn spasmodically towards the vertebral column, as in cases of painter's colic. The tongue was dry, and coated with a brown fur; the lips parched; and there was intense thirst. Under treatment the patient speedily recovered. That the symptoms in this case were not due to poison was established; 1, by their very sudden invasion in a severe form; 2, by the time which had elapsed since any food had been taken; 3, by the fact that other persons had partaken of the same food, and did not suffer. The symptoms were unlike those produced by arsenic or the common irritants; but in many respects they resembled those observed in the reported cases of ileus depending on mechanical strangulation of the intestines. The obstruction may have been caused by spasmodic constriction of the intestines; for if the cause had been mechanical, it is difficult to understand how it could have been removed. This disease sometimes assumes a chronic form. It commences with colicky pains in the abdomen, and admits of relief by the usual remedies. After a time, purgative medicines cease to act; and the abdomen becomes distended. There is then vomiting, and this speedily assumes a fæcal character. These symptoms are unlike those of irritant poisoning in any form, and an analysis of the vomited matters, or of the urine, would show the absence of poison.

Cases of obstructed (or internally strangulated) intestine have occasionally given rise to difficult medico-legal inquiries. The following is quoted by Flandin. A German physician was required to inspect the body of a merchant who had lived upon bad terms with his wife. It was supposed that she had killed him by poison. The deceased had been confined to his bed for several days, and had complained of incessant nausea, vomiting, and severe pain. A careful inspection of the body showed that a portion of the colon had become strangulated, and that it was in a gangrenous condition. The accused was immediately discharged. (Des Poisons; i, 295.) This fact, with the one which follows, proves that the abdominal viscera ought to undergo a strict examination in suspected death from poison. In February, 1829, an opera-dancer was suddenly seized with violent vomiting and obstinate constipation. There was but little pain in the abdomen, and no thirst. After two days there was a fixed pain in the right iliac region; the vomited matters were of a yellow color, and soon assumed a fæcal character. The existence of ileus was suspected, and the case soon terminated fatally. Some days after the interment of the body, reports were spread to the prejudice of the husband. The body was disintered, examined, and a statement made that deceased had died from a chronic gastro-enteritis. The legal authorities then required MM. Orfila and Rostan to make an inspection; and they found that the colon was strangulated near its junction with the cæcum by a short fatty appendage, adhering at its two ends to the mesentery, so as to form a kind of ring. There was no trace of poison. Strangulation was obviously the cause of death. (Orfila, Toxicologie, ii, 720, 1843.) The subjoined case shows that fatal strangulation may take place even before the protrusion of a hernia. A soldier, who had hitherto enjoyed good health, was suddenly seized with violent pain in the abdomen, vomiting, and obstinate constipation. He was taken to a military hospital. Severe pain was experienced on touching the surface of the abdomen, especially in the region of the navel. There was no appearance of a tumor in any part. The pulse was small and frequent, and there was intense thirst. Nine hours after admission, there was copious vomiting, with some appearance of fæcal matter, and the man died immediately afterwards. On inspection, a portion of the ileum, about the size of a small nut, was found strongly compressed in an aperture in the right inguinal canal; but there was no accumulation of fæcal matter in or near the part. The symptoms and appearances rendered it certain that deceased had died from peritonitis. (Gazette Médicale, Dec. 19, 1846, p. 995.)

Intussusception is a disease which frequently occurs in infants or children. It consists in the reception of one portion of the bowels into another. This leads to a constriction or strangulation of the portion received, and a complete obstruction of the canal. Either nothing is passed per anum, or only a small quantity of blood. It may occur in any part of the bowels; but it is most commonly observed at or near the union of the small with the large intestines. The invaginated portion of bowel varies from an inch to eight or ten inches in length.

The disease appears to result from spasm in the intestines, depending on dentition, worms, or other causes of irritation. Purgatives have been known to produce it. The chief symptoms are pain, vomiting, and convulsions: there is no purging. In one instance, this disease was mistaken for arsenical poisoning, and the mistake nearly led to the conviction of the mother and grandmother of the child, on an unfounded charge of murder. (Reg. v. Dore and Spry, Central Criminal Court, Aug. 28, 1848; also Medical Gazette, Nov, 24, 1848.)

An examination of the surface of the abdomen in any of the forms of this disease, may not always suffice to indicate the cause of the sudden illness and death. Nevertheless, the obstinate constipation, with other symptoms, will in general be sufficient to show that they cannot be ascribed to irritant poison. In these doubtful cases, if the symptoms really be dependent on poison, some connection may be generally established between the last meal taken and the period of their occurrence.

COLIC.—This disease can only be confounded with one variety of irritant poisoning, namely, that induced by the salts of lead. But it is to be observed, that the poisonous salts of lead are very rarely used criminally, and when they are taken in sufficiently large doses to kill rapidly, the symptoms resembling colic are mixed up with those of irritant poisoning,—so as to render it impossible for a practitioner to refer them to the disease alone. It is the *chronic* form of lead-poisoning which resembles colic. This is generally recognizable by the blue line on the gums, the aspect of the patient, and the history of the case.

HÆMATEMESIS.—In this disease there is neither pain nor purging; and there is a copious discharge of *blood* by vomiting. These characters show that it cannot be easily mistaken for irritant poisoning.

DISEASES RESEMBLING NEUROTIC POISONING.

We have now to consider the diseases which are attended with symptoms resembling those induced by *neurotic* poisons. They are apoplexy, epilepsy, tetanus, diseases of the brain, diseases of the heart, and rupture or distension of the stomach. Indeed, it may be remarked, that every condition of the body in which life is liable to be suddenly destroyed, from whatever cause, may be mistaken for neurotic poisoning. The various causes of sudden death should therefore be especially studied by a medical jurist. They are not very numerous, and are principally confined to diseases which affect the brain, heart, and lungs. (For an account of these causes, I must refer the reader to the Ann. d'Hyg. 1838, ii. 145; 1843, ii. 435; also to the elaborate work of Herrich and Popp, Der plötzliche Tod aus inneren Ursachen, Regensburg, 1848.) There is another point to be attended to, namely, that those fatal diseases only of these important organs, are likely to be confounded with this form of poisoning, the existence of which had not been previously suspected or announced by the usual attendant symptoms. On the trial of Tawell for poisoning Sarah Hart by prussic acid, the statistics of the causes of sudden death were entered into in the defence, in order to establish a probability that the deceased had died from natural causes, among which was placed "mental emotion!" It may be as well therefore to state, that on an average of five years, the annual number of sudden deaths in England and Wales amounted to 3600, or one in one hundred and thirty-eight of the total deaths. (Registrar-General's Report.) Although inquests were held in all of these cases, for the alleged purpose of determining the cause of death, no cause whatever was assigned in *two-thirds* of the number; and thus a vast source of knowledge of great importance in the settlement of disputed questions relative to poisoning, was entirely cut off from the profession! In a case referred to me (Sept. 1844), the jury, under the direction of a coroner, returned a verdict of death from poison ("misadventure"), while the stomach of the deceased was in my custody, and before it had even been opened, or the seals of the vessels containing it, had been broken! In another, in which there was strong reason to suspect death from poison administered by a quack, the coroner and jury declined waiting for an analysis of the contents of the stomach, although strongly advised by the medical witness who inspected the body,—and they returned a verdict of "*natural death.*" (See the case of Reg. v. Freeman, Taunton Lent Assizes, 1845.)

APOPLEXY.—Those neurotic poisons which act specially on the brain (cerebral poisons), of which we may take opium as the type, actually seem to produce this disease. The distinction of apoplexy dependent on disease, from that kind of apoplexy induced by poison, is difficult unless we can obtain a full history of the case. The following circumstances may be remembered in our diagnosis: 1. Apoplexy, as a disease, is sometimes preceded by warning symptoms before the fatal attack comes on. In poisoning, such symptoms would be wanting unless the poison were administered to a person who had already been threatened with apoplexy. 2. Apoplexy, as a disease, does not commonly attack persons under the age of thirty. The fatal cases increase progressively with age, and according to the researches of Dr. Burrows, the disease is most common between the ages of sixty and seventy. We shall presently see that there are, however, exceptions to this statement. Poisoning may be witnessed in a person at any age. 3. The relation between the time of the attack, and the time at which food or medicine was last taken. Thus if the symptoms of stupor do not come on until five or six hours after some liquid or solid has been swallowed, they are much more likely to be dependent on apoplexy from disease than on poison. This is an important character; but its occurrence is of course purely accidental, for it is by no means unusual that an attack of apoplexy should speedily follow a meal made by a previously healthy person. However, several cases have already been related, which show that this criterion may be sometimes usefully employed to distinguish disease from poisoning (ante, p. 111). 4. In apoplexy from disease, it is usually observed that coma (complete insensibility) is at once induced; but in poisoning, this symptom comes on slowly, and is generally preceded by giddiness and stupor. 5. The discovery of poison in the food taken or in the contents of the stomach;—this would at once establish the fact of poisoning. 6. The discovery of appearances in the brain indicative of apoplexy, such as effusion of blood or serum. This would negative, *cæteris paribus*, the presumption of poisoning. (See a paper by M. Tardieu, Ann. d'Hygiène, 1854, ii, p. 158.)

It is to be observed, that in all cases of disease simulating narcotic (cerebral) poisoning, the disease is assumed to prove fatal; hence, there is always an opportunity of searching for the two last-mentioned characters. We do not hear of an attack of apoplexy from which a person recovers, ever being mistaken for a case of poisoning by opium, but we hear of poisoning by opium being not unfrequently mistaken for apoplexy or convulsions. Dr. Birt Davies has published the two following cases: A person died in what was considered by the physician and surgeon attending, to be a fit; but opium was found in the stomach. A person was attended by a physician and surgeon for some hours. The illness and death were ascribed to and treated by them for apoplexy, but it was proved beyond all doubt that the deceased had died from laudanum. (Borough Inquests. Birmingham, 1845.) Such cases I am persuaded are frequent. Deaths have been registered as from "natural causes," when on an examination of the bodies some weeks or years afterwards, the deceased persons have been found to have died from poison. It is impossible to say how many of such cases escape notice for one which is brought to light. These facts show that inquests without examination of the body, by lulling suspicion, serve, in many instances, to conceal rather than to detect crime.

In reference to the age at which apoplexy may make its attack, it may be remarked that healthy girls of the respective ages of sixteen and twenty-two, have died suddenly from this disease. There had been no warning symptoms whatever. In January, 1839, a gentleman, aged twenty-two years, retired to his bedroom in good health. Shortly afterwards a servant entered the room, and found him lying on the floor dead. On an examination of the body, it was found that one of the vessels of the brain had become ruptured, and that a large quantity of blood had been effused on the surface. There was no doubt that this was a case of apoplexy in a young man who had suffered from no warning symptoms. It turned out, on inquiry, that the father and mother of the deceased had both died suddenly from the same disease.

In the following case a suspicion of poisoning was actually raised, and was only removed by a proper medico-legal examination. In September, 1838, a young healthy female, while sitting with her parents taking her supper, suddenly fell back in her chair in a state of insensibility. Medical assistance was called in, but she died in eleven hours without recovering her consciousness. Owing to her sudden death at a meal, the parents were accused by the neighbors of having administered poison to the deceased at her supper; but the medical attendant, on making an examination of the body, clearly showed that death was caused by an effusion of blood on the brain, from the rupture of a diseased bloodvessel. As apoplexy is very unusual in the young, this rather tended to strengthen the suspicion of poisoning; although it is obvious that there is no common poison which would have produced the immediate insensibility observed, except hydrocyanic acid; but when taken in so large a dose as to produce this sudden and violent effect, the patient would die in a few minutes and the poison be found in the body. No poison was discovered in this case; death was undoubtedly caused by apoplexy.

I have known a child between two and three years of age die from congestive apoplexy; and the disease has been observed to occur even in infants. Dr. A. Campbell reports a case of apoplexy proving fatal in a child only eleven days old. (North. Jour. Med. Jan. 1845.)

Some late researches on the causes of sudden death in infants have led to the result that apoplexy is more frequent than it was formerly supposed to be. A child seventeen months old was found dead in bed. An inquest was held, and an inspection of the body was made. There was no mark of violence upon it; but the face was livid. On opening the head, there was an effusion of dark-colored venous blood beneath the membranes,—the brain was softened, the vessels were congested, and there was an unusual quantity of serum in the ventricles. The other organs were healthy. In a second case, a child aged two years died under similar circumstances. The only appearance was congestion of the brain with an effusion of blood. In a third, aged nine months, the child became suddenly black in the lips and dark in the face: she drew her hands up, was convulsed, and soon afterwards died. There was some suspicion of poisoning here. On inspection, the countenance was dark: the arms were stiffened and the fingers were contracted in the palms of the hands. The pupils were dilated. There were no marks of external injury. The brain was congested and two teaspoonfuls of blood were effused at the base. The lungs were congested: the other organs were natural. There was milk in the stomach. Death was referred to effusion of blood on the brain (apoplexy). In a fourth case, a healthy child, aged fifteen months, was suddenly seized with a convulsive fit a quarter of an hour after its breakfast and died in ten minutes. The only unusual appearance was that the stomach was nearly full of food. The membranes of the brain were congested and some blood had been effused. In a fifth case, an infant two months old was found dead in bed; it had apparently died in convulsions. The brain was soft, the vessels congested, and some blood effused at the base. This effusion was the cause of death. The stomach was distended with food. It is probable that distension of the stomach may lead to an attack of apoplexy, and death from this cause may be attended by convulsions. (See Lancet, Jan. 20, 1855, p. 771.)

A remarkable case, involving the question—whether death was caused by apoplexy or prussic acid, came before the Senate of Chambery in April, 1843. I allude to that of M. Pralet (Ann. d'Hyg. xxvi, 399; xxix, 103, 474), which appears to have excited as much attention on the continent as the case of Sir T. Boughton in England. Several medical witnesses deposed that the deceased had died from prussic acid, administered to him by M. L'Héritier, the accused. Orfila was required to examine the medical evidence, and found it extremely defective. The inferences drawn from the application of the chemical tests were highly incorrect; and the results were essentially negative. Had it not been for the interference of Orfila, it is most probable that the accused would have been convicted, more from the medical opinions against him, than from the strength of the medical facts of the case. The witnesses appear to have acted on the principle, that the whole of their duty consisted in rendering the charge of poisoning probable; whereas, we shall hereafter see that no person can be convicted of this crime on mere *probability:* the fact of poisoning must be made reasonably certain, either by medical or moral evidence, or by both combined.

A medical jurist should bear in mind, that a superficial examination of the body will not suffice to reveal the cause of death on these occasions. Dr. Bell has reported a case, which is in this respect instructive. A stout, strong, and hale laborer, aged sixty-two years, who had never complained of any indisposition, resumed his work as usual after having eaten a hearty dinner, and shortly afterwards, while wheeling a barrow, he dropped down senseless, and died in a few seconds. On a careful examination of the head, it was found that fatal effusion of blood on the brain had taken place from a slight rupture in the right lateral sinus. (Med. Gaz. xxxix, 31.)

Atelectasis (imperfect distension) *of the lungs.*—It is not probable that this condition of the lungs in a new-born child would be mistaken for a case of narcotic poisoning; but in one instance, owing to the concealment of the facts, a case of poisoning by opium in an infant four days old, was mistaken for atelectasis. Dr. Balfour left the child on the previous day apparently in strong health. When summoned, he found it pulseless, and the skin cold and livid. Means were used for resuscitating it, but no intimation was made that a narcotic had been given to it. The child died in thirteen hours. On inspection, the body was generally livid, the hands were clenched, and the thumbs were drawn into the palms. The pupils were natural. The membranes of the brain were congested with dark liquid blood, and two teaspoonfuls of serum were effused at the base. The substance of the brain as well as of the cerebellum was much congested. The heart was healthy; the foramen ovale was quite open; and there was some congestion of both lungs. The stomach was healthy; it contained a thickish fluid having no odor of laudanum. Dr. Balfour remarks, in reference to this case, that there were some circumstances in which it was unlike atelectasis. Instead of being feeble from birth the child had been healthy and strong; the lividity of the surface was more persistent and more strongly marked. The spasmodic contraction of the hands was also a peculiar feature. As there were no suspicion of poisoning during life the state of the pupils was not observed. The facts were explained by the father admitting that he had given to the child two drops of laudanum to soothe it, and in about four hours the symptoms above described came on. (Edinburgh Monthly Journal, 1856–7, vol. ii, p. 148.) It will be perceived that the symptoms and appearances are similar to those described under apoplexy in infants (ante, p. 132). As no trace of so small a quantity of opium can be found in the stomach, these cases present great difficulty.

EPILEPSY.—This disease, in some of its symptoms, resembles poisoning by prussic acid only. If the symptoms depend on poison, some liquid or substance must have been taken immediately before their occurrence. If, however, nothing has been taken, the inference would be that the symptoms most probably depended on disease. Death is commonly very rapid in poisoning by prussic acid; but a first attack of epilepsy is not often fatal. If the person has suffered from previous attacks, it is probable, *cæteris paribus*, that the symptoms depend on disease. But epilepsy may, by coincidence, immediately follow the administration of a draught, or the taking of food; an analysis of the substance taken, would then remove any doubt. Supposing none of this to be procurable, then we must remember, that epilepsy only simulates narcotic poisoning when the attack is rapidly fatal. Therefore, an opportunity will always present itself for verifying or rebutting the suspicion of poisoning, by examining the contents of the stomach. I have never met with an instance, in which a case of epilepsy was mistaken for one of narcotic poisoning.

TETANUS.—When this disease occurs, it can generally be traced to some cause,—a wound, ulcer, burn, or other injury, involving tendinous or nervous structures. Tetanus may arise from causes of a very simple kind, and independently of wounds,—as from exposure to wet and cold, or to a current of air. It has been stated that it may even come on without any apparent cause. The cause may, however, have been latent. When it is the result of physical injury, it is called *traumatic;* under other circumstances, *idiopathic;* but idiopathic tetanus is by no means common; it manifests itself by trismus (locked jaw), opisthotonos, or emprosthotonos (a tensely curved position of the trunk backwards or forwards, as the result of muscular spasm). The disease occurs sometimes spontaneously in infants, within the first eight or ten days from birth (*trismus nascentium*). Male adults, especially those who are of a robust and vigorous frame, are most liable to attacks of tetanus. According to Dr. Gregory, tetanus, from cold, occurs, for the most part, within three or four days after exposure to the exciting cause; while traumatic tetanus (from wounds) generally appears about the eighth day. (Practice of Physic, 378.) Other observers have found that tetanus from wounds, very commonly shows itself from the fourth to the sixth day after the injury. The sooner it commences after an injury, the more rapidly fatal is its course.

Tetanus, or rather tetanic convulsions, may be produced by certain neurotic poisons, which affect the spinal marrow (spinal poisons), especially those belonging to the strychnos tribe,—as nux vomica—strychnia, and all its saline combinations; and there is not only a strong similarity in the symptoms, but an examination of the dead body does not indicate the existence of any well-marked morbid changes in either case. In tetanus, from disease or injury, there is a gradual progression of the symptoms. The rigid contraction commences in the muscles of the jaws; it extends to the throat, back of the neck, and, lastly, descends to the abdomen and lower limbs. Professor Colles has remarked, that the muscles of the fingers are the last and least affected. (Lectures on Surgery, i, 72.) The rigidity of the muscles continues, more or less, throughout the disease without intermission; whereas, in tetanus from poisoning, there are remissions or intervals of relaxation. A distinction will commonly rest upon the following circumstances: 1. The period of time which has elapsed since any substance, liquid or solid, was swallowed by the patient. 2. The gradual or sudden and violent accession of symptoms,—the latter indicating poisoning. In tetanus from disease, the stiffness is first perceived in the jaws; it then progressively extends downwards, attacking the body and limbs, the hands not being commonly affected until the last. In tetanus from poisoning, the attack is preceded by shivering or trembling and gasping for breath, the body and limbs are then simultaneously affected; the hands are clenched, the feet curved, and the jaw is not commonly fixed, until a late period, and during a paroxysm. 3. The duration of the case. Tetanus, as a result of local injuries, rarely proves fatal in less than twenty-four hours; and in the idiopathic form, it either does not destroy life, or only after the lapse of many hours or days. In tetanus, produced by strychnia given in fatal doses, the person rarely survives two hours after the occurrence of the symptoms. 4. The absence of any wound, ulcer, burn, or personal injury, nervous susceptibility, or exposure to cold, to account for the attack. 5. The discovery of nux vomica, strychnia, or other poison, in the food, in the matter vomited, or in the contents of the stomach after death.

The cases of Miss Abercromby (1830), and of J. P. Cook (1856), are important in reference to the distinction of the symptoms produced by spinal poisons and tetanus as a result of disease. In Miss Abercromby's case there is great reason to believe that poisoning was mistaken for disease,—a mistake much more likely to arise than the converse. Miss Abercromby was a healthy young lady in the prime of life; she was induced by her brother-in-law, Wainwright, to insure her life for two years for 3000*l.* in the Imperial Assurance Company, Wainwright having no pecuniary interest in her life. The policy was effected in October, 1830, and she died rather suddenly in December following. It was only after the lapse of five years that Wainwright brought an action against the Company for the amount of the policy (Wainwright v. Bland, Exchequer, June 29, 1835), and the evidence was such that the jury were equally divided, so that no verdict was given. The plaintiff was obliged to withdraw from the country. He died in one of our penal colonies, and, before his death, it is reported he substantially admitted that he had destroyed Miss Abercromby and several other persons by strychnia. This poison had only been discovered twelve years previously, and it was then but little known either in this country or in France.

The payment of the policy was disputed by the Company on the ground of fraud, and the defence was substantially that the lady had died from poison administered by the plaintiff. She had been for a few days indisposed with an hysterical attack, but there was nothing to excite alarm. All that could be learnt of her death was that the physician in attendance was suddenly sent for between two and three o'clock. "She was in *convulsions resembling those which were the effects of a wound* (tetanus), and said she was sure she should die, and she went off into convulsions." The physician left the house, returned at four o'clock, and she was then just dead. The appearances presented by the body are imperfectly reported. There was congestion of the vessels of the brain, with some effusion, and the bloodvessels of the stomach were distended. The cause of death was assigned to *convulsions* produced by some oysters which she had eaten for supper,—and to wet feet! The

Attorney-General put it to the jury whether it was oysters or some poison which had caused this lady's death, a point which they felt unable to decide (Medical Gazette, vol. xvi, p. 606). The cause assigned was quite inadequate to explain this sudden and rapid death. (See the case of Assistant-Surgeon Bond, post, p. 140.)

Tetanus may be the result of *hysteria*, and as such, it is chiefly met with in females, and may be traced to injury to the brain or a peculiar constitution. An attack even in a severe form may be brought on by slight causes producing mental emotion or excitement. It will probably be found on inquiry that the patient has been subject to previous attacks or fits. The spasms of hysteria may be tetanic; but convulsive motions of the limbs more commonly alternate with stiffness or rigidity, and the attack is generally attended with loss of consciousness. In poisoning, the patient retains consciousness, and the paroxysms, if frequent and severe, are generally fatal. In cases of hysteria or hysterical excitement, the attack is not fatal, but the patient speedily recovers. Such, at least, is the result of experience up to the present time.

I am indebted to Mr. Brook, formerly a pupil of Guy's Hospital, for the particulars of a case of tetanic convulsions depending on mental excitement and emotion, which from their severity might, without due precautions, have been mistaken for the effects of strychnia-poisoning. In May, 1858, he was called to a man, æt. thirty, and found him lying in bed quite insensible, his head thrown back, his body arched in a state of opisthotonos, and every joint rigid. The breathing was scarcely perceptible; the pulse was regular, and between seventy and eighty. The mouth was spasmodically closed; he was unconscious, and there was no power of swallowing. The hands were spasmodically closed; the feet curved or distorted by spasm; the ankles and wrists were relaxed, but the knee-joints could not be bent. He had been in this state about ten minutes. The muscles after a time were partially relaxed, but another paroxysm occurred, and there were three similar fits in about half an hour. After this, consciousness slowly returned, and in about an hour and twenty minutes from the first attack, the convulsions entirely ceased. During the attack the eyes were so turned under the lids, that no portion of the cornea could be seen; on a remission of the symptoms, when the eyes were opened, the pupils were found in their natural state. The patient felt an inclination to vomit, but nothing escaped from the mouth but a bloody mucus and saliva. So soon as he could speak, he said he was subject to these attacks and would soon be better. The following history of the case was obtained. The man was an officer in the French navy, but a political refugee in this country. About four years previously, while at Calcutta, he received a severe wound in the forehead from a decanter which was thrown at him by a companion. He was very ill for some days; and shortly after receiving this injury, and up to the present date, he had been subject to frequent attacks of tetanic convulsions, generally as a result of great mental emotion or excitement. This last attack had been brought on by a quarrel with a female with whom he cohabited. He had a full knowledge of the access of the fits. They were preceded by a sense of fulness in the head, flushed face, tingling at the extremities of the fingers, and (to use his own expression) a feeling as if his heart was in a vice; with a cold perspiration.

In this case there was a natural cause for the tetanic convulsions, and this was only one of numerous attacks. In the sudden access, and in the severity of the symptoms, there was a resemblance to symptoms of poisoning by strychnia; but they differed from these in the mode of attack, the total unconsciousness, the state of the eyes, the pulse, and the breathing, as well as in the rapid subsidence of the fits, and the recovery of the patient. There had been a cause of excitement immediately before their occurrence. The effects produced on the nervous system as a result of hysterical excitement or mental emotion do not admit of postponement. The tetanic attack comes on at once or not at all.

The case of J. P. Cook (Reg. v. W. Palmer, Central Criminal Court, May, 1856) falls under the distinctive criteria above pointed out. There was no wound or personal injury. There was no reason to suppose that the tetanic convulsions from which the deceased suffered were of the idiopathic kind, *i. e.* that they had arisen from exposure to wet or cold, or from excitement, as a result of his having won a race a week before the attack. Deceased had had some pills administered to him by the prisoner, at a time which would correspond to the interval that precedes the action of strychnia. The symptoms were sudden and violent, developed over the entire body and limbs in a few minutes, and they proved fatal in *twenty minutes!* The pills could not be obtained for analysis, and no strychnia was found in the stomach, which had been cut from end to end and the contents lost under the superintendence of the prisoner. The physiological and pathological evidence, however, that deceased had died from strychnia was considered to be conclusive, and on this evidence the prisoner was very properly convicted and executed. (See Guy's Hospital Reports, October, 1856, On Poisoning by Strychnia.)

So much attention has been directed, by reason of the medical evidence given at this trial, to the subject of tetanus, that some additional remarks may serve to aid the practitioner in forming an opinion in future cases. When the circumstances of a case are fully known, there can be no difficulty; but if professional men will base their opinions upon false or partial statements, imperfectly observed, or concocted purposely to fit a defence, they must be misled. Up to the year 1856, there had been a large number of fatal cases of tetanus; the facts had been accurately recorded, the mortality noted, and no one had thought of analyzing the contents of a stomach in these cases, for the simple reason that the labor would have been superfluous. On the other hand, it is by no means improbable that some poisonings by strychnia may have been passed over as idiopathic tetanus; and of this, in my opinion, the case of Miss Abercromby (ante, p. 135) furnishes a striking example. But if medical men are to be calumniated for the honest expression and maintenance of an opinion afterwards proved to be true by the verdict of a jury, society must be prepared for the concealment of these cases. It is certainly desirable not to encourage false charges, but it is a totally different question to allow or encourage iniquitous defences. In Wooler's case (ante, p. 108) the medical men were charged with poisoning their patient, and then in order to screen themselves, imputing the act of poisoning to the husband, who was on his trial for the crime. In the case of Palmer, although Dr. Rees and myself escaped this imputation, we were charged with "incredible rashness" and "unheard-of indiscretion," because we had honestly and fearlessly given, according to our consciences, an opinion which was subsequently supported by the evidence of Brodie, Christison, Todd, Curling, Solly, and others! The great point of contention in this case was: Did the tetanic symptoms under which the deceased died, depend on disease or poison? All those authorities, comprising the most experienced men in the kingdom, agreed, that when taken as a whole, they were not in accordance with any known form of disease, but were in perfect accordance with the effects of strychnia. The opinions given by some witnesses in the defence were to the effect that the symptoms were consistent with strychnia-poisoning; and although some professed to perceive differences, the records of medical experience proved that these differences existed only in their own imaginations. In short, there was no real difficulty in this case, except that which was temporarily created by a suppression or misrepresentation of the real facts, and the sympathy excited in the public mind, by reason of the social position of the accused.

The following case of tetanus, which occurred at the London Hospital in March, 1856, was considered at the time to present a difficulty to medical opinions based on symptoms. A man aged thirty-seven, suffering from chronic indurated ulcers, two at the back of the right elbow near the ulnar nerve, and one on the left elbow, was brought into the hospital, at 7·30 P.M. He was then breathing quickly; the jaws were closed; he was unable to swallow; and the muscles of the abdomen and back were somewhat tense. In ten minutes he had a paroxysm with opisthotonos, which lasted about one minute; he was then quiet for a few minutes; he had another fit, in which he died, having been in the hospital only half an hour. It appeared that the symptoms of stiffness in the jaws had come on about *nine hours* before his admission. The surgeon who attended the case had no doubt that the man had died from tetanus, and the cause of the tetanus was referable to the indurated chronic ulcers. In fact, there was nothing in the case to lead to the supposition that the symptoms were caused by strychnia; and no treatment was adopted for poisoning by strychnia. In testing this case by the rules laid down for a distinction, we find the following differences: 1. There was no evidence that the man had swallowed anything, or had been able to swallow anything, from the time at which he had had his dinner,—eight or nine hours before his admission. There was no reason to suspect that he had knowingly or accidentally taken strychnia, or that his wife had given it to him, even assuming that this poison could have been in his body eight hours without causing symptoms and death. 2. The convulsive symptoms commenced and progressed slowly, so that eight hours had elapsed before they acquired great severity, as manifested by opisthotonos. They began, as in most cases of traumatic tetanus, with stiffness, followed by spasm and fixedness of the jaws; the abdomen, back, and extremities being only long afterwards affected;

there was inability to swallow; and although the arms and legs were convulsively affected in the paroxysms preceding death, it does not appear that there was any clenching of the hands, or incurvation of the feet. 8. From the date of the commencement of symptoms (in the jaws) the man lived eight or nine hours. Apart from the absence of all moral evidence, there was not a single feature in the commencement, progress, and duration of the symptoms in this case, by which it could be confounded with a case of strychnia-poisoning. Although more rapidly fatal than traumatic tetanus is usually observed to be, it would have been a case of uncommon duration for one of strychnia-poisoning. In tetanus from disease, the case is reckoned by hours or days, in tetanus from strychnia, by minutes. An analysis of the contents of the stomach of deceased was formally made by Dr. Letheby, although there was not the slightest ground for suspecting death from poisoning. No strychnia was found, where none could have been swallowed; but the case was brought forward in the defence of William Palmer, apparently for the purpose of showing that an expert of Dr. Letheby's experience might be easily deceived, and that tetanus from natural causes, except by careful chemical research, could not be distinguished from that produced by strychnia! I need hardly observe that but for the peculiar circumstances under which Dr. Letheby was then placed (his services having been specially retained for the defence of Palmer), he might probably have been inclined to view this case as experienced surgeons viewed it at the time, and at the trial, namely, as a simple case of tetanus caused by chronic indurated ulcers in the region of a nerve; and that a chemical analysis was not required.

Whenever the symptoms of tetanus appear suddenly with severity, and the patient dies, a minute investigation should be made into the history of the patient, and all the circumstances attending the attack. In traumatic tetanus the cause is often overlooked,—a small splinter of wood or metal may penetrate the palm of the hand or sole of the foot,—the wound may completely heal over the foreign body, and no suspicion of latent mischief may exist. In the spring of 1858, a man was admitted into Guy's Hospital, suffering under tetanus; he died in three days with the usual symptoms. The cause of the tetanus was traced to a wound in the hand received a *month* before; this was perfectly healed, and from the length of time which had elapsed it was not thought of importance, but on cutting into the wounded part after death, a piece of rusty iron was found imbedded therein, and pressing on a nerve. In another case, a young man was admitted, and died from tetanus in ten hours. On inspection, it was found that a slight wound in the hand, received shortly before, had completely healed and inclosed a splinter of wood,—the exciting cause of the disease. The facts connected with these cases were not consistent with the theory of poisoning by strychnia, nevertheless, they show that after the complete healing of a wound, and at a long interval, traumatic tetanus in a fatal form may insidiously make its appearance. (See, for another case in which the cause was mistaken, Medical Jurisprudence, 6th edit. 1858, p. 335.)

On the other hand, poison may have been unconsciously taken, and the symptoms referred to disease. This occurred in the case of Assistant-Surgeon Bond, at Moulmein (March, 1858). This gentleman, intending to take two aperient pills, swallowed by mistake two pills containing in each one grain of strychnia. He was seized with violent tetanic convulsions and opisthotonos, and died in less than *two hours* after the commencement of the symptoms. Deceased was unconscious of having made a mistake in taking his medicine, even to the last, and his medical friends had at the time no suspicion that strychnia was the cause of the symptoms. His illness was attributed at first to irritation of the spinal cord proceeding to inflammation, arising from his having been exposed to a current of cold air while in a heated state. It was not until after he had expired, that it was found that he had taken pills containing strychnia in place of aperient pills. I am indebted to his medical attendant for this information. But for the accidental discovery of the strychnia pills, the tetanus might have been referred to hysterical excitement, or some "undiscoverable disease" of the spinal marrow, although its characters were clearly those of strychnia-poisoning. A case reported by Dr. Lonsdale (Edinburgh Monthly Journal of Medicine, Feb. 1855, p. 117) shows the dangerous facility with which tetanus, as a result of poisoning by strychnia, may be overlooked. In Nov. 1854, a man, æt. fifty-nine, went, apparently in his usual health, early in the morning, to bathe in the river Esk, near Carlisle. About eight o'clock he was seen walking home, and on reaching his own house he complained of severe illness, was violently cramped, and declared himself dying. A doctor was immediately called, who prescribed a pill, but the man after exhibiting some marked symptoms, died within thirty or forty minutes of his arrival at home. As deceased had often suffered from inward complaints,—was a frequent patient at a County Charity, and had that morning (in November) taken an early bath in the river, it was considered by his neighbors that the cold had struck him, and his disease was looked upon as sudden death from natural causes! An inquiry before the coroner, however, led to the discovery that the man had been accidentally poisoned by strychnia. The physician who saw deceased during his illness stated, that when called to him at half-past eight A.M., he was laboring under violent spasms, which almost entirely subsided in from four to seven or eight minutes. During the spasms the body was extended, with the limbs separated, stiff, and rigid, and there was a violent shaking of the whole body. At first the spasms were most marked down the back and legs, but in the course of from ten to fifteen minutes they fixed upon the chest, and violent tetanus supervened with fixation of the muscles of respiration, and in this state the patient died. Deceased was anxious, agitated, and felt certain of impending death. His intellect was perfectly clear. The eyes protruded, the pupils were dilated, and the mouth was spasmodically closed. The face and hands were livid, and the surface below the natural temperature. It turned out, on due inquiry, that on his return from the river, deceased had called at a druggist's for a strong dose of purgative medicine, and the druggist, as it was afterwards shown, had served him with a grain and a half of strychnia by mistake for jalapine! The symptoms and death had been caused by poison,—not by natural disease.

Dr. Roberts has reported a case in which tetanic convulsions of a severe kind attacked a man as a result of exposure to cold. They are said to have resembled those of strychnia-poisoning in coming on suddenly, in their great violence, in affecting the limbs as well as the trunk, in the early production of opisthotonos, in the retention of consciousness, and in the existence of intervals of relaxation between the paroxysms. After seven hours, the man recovered. It appears that he had a feeble constitution, as well as a nervous temperament, and he stated that his feelings were easily excited. He had lately suffered much from mental anxiety, and had, moreover, been exposed many hours to severe cold. (Lancet, March 27, 1858, p. 313.)

These three cases show the very difficult position in which a medical jurist may be placed. On the one hand, he may assign to poison, symptoms which are really caused by disease; on the other, he may be induced, from an imperfect knowledge of the facts, to refer to disease, a death which is actually caused by poison. In the latter case, he not only lends his science to conceal a murder, but he advertises a method by which a number of lives may be easily sacrificed, and criminals escape with impunity! In Dr. Roberts's case, the man had taken nothing for four hours before the symptoms set in; there had been no possession of, or access to strychnia,—there had been long exposure to a benumbing cold in a person exhausted by destitution and fatigue; the convulsions lasted at intervals for seven hours, becoming less and less violent, and then disappeared, and lastly, the man *did not die*. It is stated that there were no suspicious circumstances in the case; and the man was preternaturally susceptible of a convulsive attack. If under this state of things a medical practitioner had assigned the symptoms to poisoning by strychnia, his conduct would have been justly open to censure. Each case must be decided by *all* the circumstances, medical and moral, which attend it. An implicit faith in a few symptoms will expose a man to the risk of setting free a murderer, or of leading to the conviction of an innocent person. It is proper to remember on such occasions, that a charge of murder by poison is not likely to be raised unless there are "suspicious circumstances," and unless death takes place suddenly with violent symptoms, in the entire absence of any apparent or probable natural cause. In the cases of Assistant-Surgeon Bond, and of the man whose death is reported by Dr. Lonsdale, there was *prima facie* evidence for believing that these persons had died of poison. In Dr. Roberts's case there was an absence of such evidence. As cholera may sometimes put on the features of poisoning by arsenic, so may tetanus occasionally put on the features of poisoning by strychnia. In either instance a close sifting of all the facts is necessary before we can form a correct medical opinion; the exceptional resemblance furnishes no reason for abandoning every future case as unsolvable by medical science, and thus giving free scope to secret murder in its worst and most dangerous form.

The remarks above made have been chiefly restricted to poisoning by strychnia; but tetanus or tetanic convulsions may be an effect of other poisons. Arsenic, tartarized antimony, and prussic acid have been known to produce them. In these cases, however, the tetanic are either preceded or followed by other symptoms of a special kind, which remove any difficulty in the formation of an opinion. The presence of poison in the vomited matters during life, or in the contents of the viscera after death, will also aid a medical practitioner in forming his opinion.

Convulsions.—This is a frequent cause of death among children. According to the Seventh Report of the Registrar-General (1846), the yearly average of deaths ascribed to this cause then amounted to 25,000. Convulsions (or fits, as they are vulgarly termed) may arise from the action of poisons, especially of those belonging to the neurotic class, or from the effects of disease. As they sometimes attack children suddenly, and prove fatal rapidly, a suspicion may arise that death has been caused by some poison administered to the child. Many cases of this kind have been referred to me for investigation; and, from the frequency of their occurrence, and the unjust suspicions to which they may give rise, it is the duty of a practitioner to make himself acquainted with the ordinary causes of convulsions. Medical writers have divided them into symptomatic and idiopathic. They are most commonly symptomatic, *i. e.* depending on some disease or morbid condition of the system,—such as dentition, repelled eruptions of the skin, water on the brain, exposure to cold, indigestion, worms, accumulation of fæces, improper food, or over-distension of the stomach and bowels with food; and even a peculiar condition of the nurse's milk may become a cause. The younger and more irritable the child the greater is its liability to an attack; and in these instances, the slightest cause of irritation to the nervous system may lead to it. About the time of the appearance of the first set of teeth, *i. e.* from the fifteenth to the eighteenth month, children are considered to be most liable to the disease.

When the convulsions cannot be traced to any of the causes above assigned, they are described as idiopathic, and are commonly referred to some primary disease of the brain; and this organ, after death, may be found in a state of congestion. Idiopathic convulsions sometimes run through their course and cause death very rapidly: but it is not at all improbable that, by diligent inquiry, some cause may be generally found. Dr. Underwood met with several instances in which fine healthy children died suddenly from convulsions soon after they had been overfed by their nurses. This is, no doubt, a common cause of death in infants (ante, p. 132).

Convulsions are a very common effect of the action of overdoses of opium on children; and they are not easily distinguished from those which arise from natural causes. During the fit the eyes are distorted, the pupils contracted or dilated. The spasm may affect the organs of respiration: the jaws are closed, and saliva, in a frothy state, escapes at the mouth. There may be also stertorous or snoring breathing; and, from impeded respiration, the tongue, face, and the surface of the skin will become livid, owing to imperfect aeration of the blood, and the child may die asphyxiated. Under prompt and appropriate treatment, except when it depends on poison unsuspected, the attack may be alleviated, and the child recover. When a neurotic poison is the cause, it will be found that some substance, either liquid or solid, has been given to the child not long before; and the treatment to be pursued in a suspicious case must be directed to its removal. (See Opium.) Except by a chemical analysis of the food and the contents of the stomach, it is by no means easy to distinguish disease from poisoning.

A case was tried at the Lincoln assizes, which shows that a crafty criminal may easily deceive a medical practitioner, and that an inquest, as it is at present frequently conducted, is not fitted to detect secret cases of poisoning. In this instance a confession was made; but how many instances escape detection for want of a confession on the part of a criminal, it is impossible to conjecture. An inspection of a body is not required by many coroners unless there are strong circumstances for suspicion in the shape of public rumor; but in respect to criminals, who have well calculated their plans, these circumstances are not likely to come to light except from the inspection itself and an analysis of the contents of the viscera. Coroners are not always to be blamed for the omission to require proper medical evidence. They are thwarted on many occasions by county magistrates, who frequently disallow their fees, even when the expense of the inquiry has been necessary to a proper verdict. It does not appear that an inquisition was held, or inspection made, in the case alluded to, until some time after the bodies of the deceased had been interred, and then it was too late. A woman was charged with the murder of three children, by poisoning one of them with arsenic, and the other two with opium. She pleaded guilty, and described the mode in which the crime was perpetrated. She had succeeded in poisoning two of the children without being detected; although suspicion was so strong that she was tried, but acquitted, at the previous assizes, on the charge of having poisoned one of them. In the third case, she admitted having secretly given to the deceased (her own infant), about three weeks old, a teaspoonful of laudanum. The child was soon afterwards seized with convulsions; a medical practitioner was sent for, who, deceived by the statement of the woman, treated it as a case of ordinary convulsions in children, and ordered a warm bath. The child died in about twenty hours, continuing, according to the prisoner's statement, in convulsions during the greater part of that time. No suspicion appears to have been entertained of the real cause of death, and the case would probably have remained undiscoved, but for the prisoner's confession. It is remarkable that this child survived so long; the woman however, prevaricated as to the quantity of laudanum which she gave it, —hence it is difficult to draw from her statement any other conclusion, than that the deceased was actually poisoned by opium. (The Queen v. Joyce, Lincoln Aut. Ass. 1844.)

In a case which occurred in December, 1846, a suspicion arose that two children had been poisoned, from the singular fact that they died within a very short period of each other. One, an infant, aged seven months, was found by the mother at 6 A.M. in a fit. It was livid in the face, frothing at the mouth, and its limbs were drawn up and rigid. She immediately took it to a person living in the same house, but it remained insensible until it died about two hours afterwards. The child appeared well when put to bed on the previous night, and had had its last meal (boiled bread and milk) about 7 P.M. The chief appearances on inspection, were congestion of the brain and lungs; there was slight redness of the stomach. On the same morning and at about the same time, the other child, aged fifteen months, was found by the mother insensible, dark in the face, and struggling for breath. The child died five minutes after it was found in this state. On inspection, the only appearance was general congestion of the brain. The stomachs and their contents, as well as a portion of the food given to the children the night before, were examined for opium as well as other poisons which were likely to have occasioned the symptoms; but no trace of poison could be found. There was no moral evidence to show that poison had been given; none was detected in the food; and had it been given by the mother, who found the children dying early in the morning, it is probable that, as there had been no vomiting, and death was rapid, the poison would have been discovered by the odor, either in the stomach or its contents, by the usual tests. The opinion which I gave was, that death had resulted from convulsions, probably produced by a congested state of the brain. The most remarkable feature in this case was the coincidence in seizure and the time of death; and, but for the good character of the parents and the results of a chemical examination of the food and of the viscera, it would have been difficult to have satisfied the neighbors that the children had not been destroyed by poison. The jury returned a verdict of death from natural causes. It is not enough to assign death to convulsions; the cause should if possible be indicated. The convulsions may really have arisen from some poison administered to a child: whatever affects strongly the nervous system of a child, may bring on an attack of convulsions which may prove fatal. In Reg. v. Connell (Cent. Crim. Court, Nov. 1852), the prisoner, a female servant, gave a quantity of sulphuric acid to an infant. Owing to the local effect of the acid the child was not able to take food; it became very weak, and died on the sixteenth day from convulsions. The medical witness assigned this fatal attack to the poison, because the child had not been before subject to fits, and the cause appeared inadequate. It was admitted that convulsions might arise from other and natural causes, and the prisoner was acquitted.

DISEASES OF THE BRAIN AND SPINAL MARROW.—Among these diseases may be mentioned inflammation of the brain and its membranes, hypertrophy, and the formation of tumors. Such diseases are of a very insidious nature: they sometimes give no warning of their presence, until the person, who may be in his usual health, is suddenly seized with stupor, followed by coma, or paralysis, and rapidly dies. All such cases resemble poisoning by opium: they can only be distinguished by the discovery of the affirmative characters of disease, on an examination of the body, and an absence of poison from the stomach. But the period of access of the symptoms after a meal, and the rapidity of death, will in many instances allow a practitioner to form a satisfactory distinction. This subject has been elsewhere considered (ante, p. 112). The following may serve as an additional illustration. A woman, aged 37, died suddenly, soon after having taken her breakfast. On an examination of the body, there was found effused within the cranium a large quantity of bloody serum; and the brain and its membranes were much congested with blood. No poison was discovered in the stomach, and it is certain, that had death been due to a narcotic, some traces of it would have been found, in consequence of the great rapidity with which the deceased died. The only points in which this resembled a case of poisoning, were in the invasion of the symptoms soon after a meal, and their rapidly fatal termination. (See Annales D'Hyg. 1854, ii, p. 160.)

DISEASES OF THE HEART.—The heart is subject to many diseases, which present the same insidious characters as those of the brain. Thus they may remain for a long time latent, and then suddenly destroy life. They are only likely to be confounded with poisoning by prussic acid, owing to the rapidity with which death takes place. In all these cases, therefore, if the fatal attack occur suddenly some hours after food or medicine has been taken, there can be no reason for attributing it to poison. It is only when by a coincidence the symptoms appear immediately after something has been swallowed by the patient, that any doubt of the cause to which they may be due can arise; and here, the doubt would be speedily removed by an examination of the body. We must not expect, however, that in these fatal affections of the heart, well-marked appearances will be always found. Some pathologists have described a singular condition of this organ, under which a person is liable to die suddenly after experiencing nausea and giddiness. In such cases, the muscular substance of the heart has been found only preternaturally flaccid, and its cavities empty. This has been called by Mr. Chevalier *Idiopathic asphyxia*, and others have termed it *Syncopal asphyxia.* It does not appear to be very common, for very little is known concerning it, or on what the cause of death really depends. In regard to its recognition in suspected cases, all we can say is, that if poisoning be not clearly negatived by concurrent circumstances, its usual affirmative characters are entirely wanting. The emptiness of the heart appears to be the chief indication of this variety of sudden death. This emptiness of the organ, however, is met with in other cases. It has been frequently observed in death from strychnia; and has here been supposed to depend on spasm of the organ. It is probable, however, that in all these cases there is a slight action of the heart in the act of dying, by which its cavities are emptied after the vessels of the lungs have ceased to transmit blood. In the case of Lord George Bentinck (Sept. 1848), emptiness of the heart was the only remarkable appearance found in the body. The deceased had died suddenly while taking a walk. He had not partaken of food for many hours. He was found dead lying flat on his face, and both of his hands were under him: his stick was firmly grasped in one hand. The body had been lying exposed five or six hours, and there was only the usual rigidity of death. At first it was suspected he had died from poison, but there was nothing to support this view. It was probably a sudden attack of syncopal asphyxia which had caused his death while in the act of walking.

The question, whether death has arisen from poison or from malformation, or disease of the heart, occasionally presents itself to a medical jurist. The distinction is of some importance, since the local changes in the organ may be slight and easily overlooked. In July, 1844, a woman, aged 45, fell suddenly in the street, near Guy's Hospital, and died, gasping and pallid, without being at all convulsed, in ten minutes after she had been brought into one of the wards. It was ascertained that she was a woman of irregular and intemperate habits, and that, about an hour before, she had purchased a drachm of powdered nux-vomica at a druggist's. A paper containing the poison was found in her possession. There was no certain evidence that she had taken any of it; but unfortunately the quantity found upon her was not weighed. It was considered, however, by those who saw it, to be equal to about a drachm, the quantity purchased. Owing to the report that poison had been taken, the stomach-pump was used. On inspection, eighteen hours after death, it was remarked that the lips and ends of the fingers were extremely blue. The heart was enlarged, the right cavities being very capacious, and distended with dark semifluid clots of blood. There appeared to have been originally two communications between the auricles. The pulmonary artery was of extraordinary width, while the aorta was narrow. The lungs were distended with air and congested; the liver congested; the mucous membrane of the stomach slightly corrugated, and it presented a few spots of effused blood, which were probably produced by the stomach-pump. I examined the stomach and its contents, but no trace of nux-vomica or any other poisonous matter was found. Although it is possible that some of the poison might have been taken, and afterwards removed by vomiting or the stomach-pump, the symptoms and the short duration of the case showed that death could not be referred to it. There was no doubt, that this was caused by a delay to the passage of the blood through the right cavities of the heart, from congenital malformation of this organ. It was ascertained that the deceased had been always liable to shortness of breathing, and that she became blue upon any unusual exertion. (See Medical Gazette, xxxvi, p. 19.)

In January, 1846, a man, aged 37, who had been for some time ailing and in low spirits, went out on his avocations, after having taken his breakfast as usual, and was observed to fall suddenly in the street. This was between three and four hours after he had taken his breakfast. He was carried into a shop; some stimulant was given to him, but he died before medical assistance arrived, which was about ten minutes after the seizure. It was ascertained that he had been kept in the erect posture after the attack. The chief appearances found in the body, were great enlargement of the heart, with a thinning of the substance of the left ventricle. The mucous membrane of the stomach was slightly reddened, and there were two small patches of ulceration,—the ulcers being thickened around the borders. The mucous glands were also much enlarged. The liquid found in the stomach contained no trace of poison. Considering that the severe symptoms commenced long after any article of food had been taken, and that, had they arisen from any poison subsequently taken, it must, from the rapidity of death and the absence of vomiting, have been found in the stomach, it was inferred that death had not been caused by poison. The opinion given at the inquest was, that death had taken place from a diseased condition of the heart producing fatal syncope. (See cases, ante, p. 145.)

DISTENSION OF THE STOMACH.—This is by no means an unfrequent cause of sudden death; it may occur in infancy, or at any age. In some instances, the distension of this organ appears to act by inducing apoplexy, the usual marks of that disease being found in the brain (see ante, p. 132). In other cases, death appears to be due to a fatal impression analogous to shock, arising simply from the excessive mechanical distension of the organ; it is not surprising that a suspicion of poisoning should occasionally arise under such circumstances. I have known several instances which have occurred in this metropolis, where the individuals went to bed in their usual health after eating a full supper, and were found dead on the following morning. On dissection, no marked changes were discovered, excepting, in some cases, slight congestion of the cerebral vessels. The most striking appearance was the enormously distended state of the stomach itself. In December, 1839, a girl, aged 22, after eating a hearty supper, retired to rest. In about two hours she was found insensible, and she died in the course of a few minutes. There was no examination of the body; although it is difficult to understand why, without it, there should have been any inquest,—as the cause of death, which was probably due to the distension of the stomach, was still left unexplained. In April, 1841, a man, aged 34, ate a full breakfast, consisting of three-quarters of a pound of beef, with bread, and a pint and a half of coffee. In a few minutes afterwards, he sat on a barrel to rest himself, but almost immediately fell backwards and expired. This cause of death may be met with in persons of all ages. In November, 1842, a girl, aged 18, ate a full breakfast; and about an hour afterwards she became insensible, and died in the course of a short time. The only cause which could be assigned for her death, was, over-distension of the stomach with food, probably leading to apoplexy.

Rupture of the stomach has been observed to occur sometimes as a consequence of over-distension, combined with efforts at vomiting; although in other instances the rupture has taken place when there was but little food found in the stomach. Death is, of course, a speedy consequence of this accident: hence no difficulty can arise in practice with regard to it, because an examination of the body would enable a practitioner at once to determine the cause. (For a fatal case of this kind, in which there was no apparent disease of the organ, see Medical Gazette, ii, 182.)

Rupture of the gall-bladder and gall-ducts, as well as of the impregnated **uterus** or its appendages, may also suddenly give rise to alarming symptoms of a suspicious kind in a previously healthy person. Death in such cases commonly takes place from peritonitis. The rules for forming a diagnosis are similar to those already described; an examination of the body suffices to develop the real nature of the case.

When called to examine a case of suspected narcotic poisoning, and the symptoms have occurred soon after a meal, a practitioner must remember that although a full meal is a very common exciting cause of apoplexy, this is not the case with any simple medicine, liquid or solid, which may have been swallowed by the patient. Should the symptoms follow the taking of a draught or any kind of medicine, the circumstances become much more suspicious; because the occurrence of apoplexy in such a case would be a pure coincidence: all we can say is, that it may happen—in proof of which we may refer to the case mentioned ante, page 111, and then we require other circumstances to aid our judgment. In such cases, it can never be assumed that the medicine taken was the cause of the symptoms, unless we suppose it to have been a poison; while when the symptoms follow an ordinary meal, apoplexy may be a natural result,—at least it is not absolutely necessary, in order to account for them, to suppose that the food contained any poison.

In reference to poisoning in the living, it may be useful to suggest to a medical practitioner those points to which his attention should be especially directed. They are: 1. The time of the occurrence of *symptoms*—their nature. 2. The exact period at which they were observed to take place after a meal, or after food or medicine had been taken. 3. The order of their occurrence. 4. Whether there was any remission or intermission in their progress, or whether they continued to become more and more aggravated until death. 5. Whether the patient had labored under any previous illness. 6. Whether the symptoms were observed to recur more violently after a particular meal, or after any particular kind of food or medicine. 7. Whether the patient has vomited: the vomited matters, if any (especially those *first* ejected), to be procured: their odor, color, and acid or alkaline reaction noted,—as well as their quantity. 8. If none be procurable, and the vomiting have taken place on the dress, furniture, or floor of the room, then a portion of the clothing, sheet, or carpet may be cut out and reserved for analysis: if the vomiting have occurred on a deal floor, a portion of the wood may be scraped or cut out: or if on a stone pavement, then a clean piece of rag or sponge, soaked in distilled water, may be used to remove any traces of the substance. [Some years since, an animal was poisoned by arsenic. None of the poison could be detected in the stomach, but it was easily found in a portion of deal floor, rendered humid by the liquid matters which the animal had vomited during the night.] The vessel in which vomited matters have been contained will often furnish valuable evidence, since heavy mineral poisons fall to the bottom, or adhere to the sides of the vessel. 9. If vomited matter cannot be procured, then it will be proper to collect the urine which may have been voided since the occurrence of symptoms. 10. Endeavor to ascertain the probable nature of the food or medicine last taken, and the exact time at which it was taken. 11. Ascertain the nature of *all* the different articles of food used at a meal. 12. Any suspected articles of food, as well as the vomited matters, to be as soon as possible sealed up in a clean glass vessel, labelled, and reserved for analysis. 13. Note down in their own words all explanations *voluntarily* made by parties present, or who are supposed to be concerned in the suspected poisoning. 14. Whether more than one person partook of the food or medicine: if so, whether all these persons were affected, and how. 15. Whether the same kind of food or medicine had been previously taken by the patient or other persons without ill effects following.

CHAPTER X.

ON THE EVIDENCE OF POISONING IN THE DEAD BODY—PERIOD AT WHICH POISONS PROVE FATAL—CHRONIC POISONING—ACCUMULATIVE POISONS—APPEARANCES PRODUCED BY THE DIFFERENT CLASSES OF POISONS—REDNESS OF THE MUCOUS MEMBRANE—MISTAKEN FOR INFLAMMATION—ULCERATION AND CORROSION—SOFTENING—PERFORATIONS OF THE STOMACH—OF THE INTESTINES FROM DISEASE—BY WORMS—SPECIES OF WORMS FOUND IN THE BODY.

SUPPOSING that the person is dead, and we are required to determine whether the case be one of poisoning or not, we must, in the first instance, endeavor to ascertain all the particulars which have been discussed in the last chapter, as indicative of poisoning in the living subject. Should the deceased have died from poison, the circumstances of the attack, and the symptoms preceding death, ought to correspond with the characters already described; and in these investigations it is well to bear in mind the following rules: There is no one symptom or pathological condition which is peculiar to poisoning; but at the same time there is no disease which presents *all* those characters that are met with in a special case of poisoning. The additional evidence to be derived from the death of the person may be considered under the following heads:

1. THE TIME AT WHICH DEATH TAKES PLACE AFTER THE FIRST OCCURRENCE OF SYMPTOMS.—This question it is necessary to examine, because the more common poisons, when taken in fatal doses, generally produce their fatal effects within certain periods of time. By an attention to this point, we may, in some instances, be enabled to negative a charge of poisoning, and in others to form an opinion of the kind of poison which has been taken. In a court of law, a medical practitioner is often required to state the usual *period of time* within which poisons prove fatal. It is to be observed, that not only do poisons differ from each other in this respect, but the same substance, according to the form or quantity in which it has been taken, will differ in the rapidity of its action. A large dose of strong prussic acid, *i. e.* from half an ounce to an ounce, may destroy life in less than two minutes. In ordinary cases of poisoning by this substance, a person dies, *i. e.* all signs of life have commonly ceased, in from ten to twenty minutes;—if he survive half an hour, there is some hope of recovery. In the case of the seven Parisian epileptics, accidentally poisoned by an equal dose of prussic acid prescribed medicinally, the first died in about twenty minutes, the seventh survived three-quarters of an hour. (See post, PRUSSIC ACID.) Oxalic acid, one of the most energetic of the common poisons, when taken in a dose of from half an ounce to an ounce, may destroy life in from ten minutes to an hour; if the poison be not perfectly dissolved when swallowed, it is a longer time in proving fatal. The strong mineral acids, in poisonous doses, destroy life in about eighteen or twenty-four hours. Arsenic, under the form of arsenious acid (white arsenic),

operates fatally in from eighteen hours to three or four days. It has, however, in more than one instance, killed a person in two hours; although this is by no means common. Opium, either as a solid or under the form of laudanum, commonly proves fatal in from six to twelve hours; but it has been known, in several instances, to destroy life in less than three hours;—those who survive the effects of this poison for twelve hours are considered to stand a fair chance of recovery. Strychnia has proved fatal in from twenty minutes to six hours after the poison has been taken. This must be understood to be merely a statement of the average results, as nearly, perhaps, as we are warranted in giving an opinion; but a medical jurist will of course be aware that the fatal period may be protracted or shortened, according to all those circumstances which have been elsewhere stated to affect the action of poisons (ante, p. 109).

There are various forms which this question may assume in a court of law;—the death of a person alleged to have taken poison, may have occurred too rapidly or too slowly to justify a suspicion of poisoning. The following case may serve as an illustration: A woman of the name of Russell was tried and convicted at the Lewes Summer Assizes, in 1826, for the murder of her husband, by poisoning him with arsenic. The poison was detected in the stomach; but the fact of poisoning was disputed by some medical witnesses, for this among other reasons,—that the deceased had died *three* hours after the only meal at which the poison could have been administered to him. The authority of Sir A. Cooper and others was cited to show, that, according to their experience, they had never known a case to prove fatal in less than seven hours. This may well have been; but, at the same time, there was sufficient authority on the other side, to establish that some cases of arsenical poisoning had actually proved fatal in three or four hours. So far as this objection was concerned, the prisoner was very properly convicted. On the medical question raised at this trial, it may be observed, that two distinct cases have occurred in which the persons died certainly within two hours after they had taken this poison; and several instances have been reported since the trial, in which death took place in from three to four hours after the administration of arsenic. It seems extraordinary in the present day, that any attempt should have been made by a professional man to negative a charge of criminal poisoning upon so weak a ground as this; but we must remember that this opinion was expressed many years ago, when the subject of poisoning, in its medical relations, was but little understood. It is quite obvious that there is nothing, so far as we know, to prevent arsenic from destroying life in an hour. These matters can only be settled by a careful observation of numerous cases, and not by any à priori reasoning, or reference to a limited personal experience.

In all instances of *sudden death*, there is generally a strong tendency on the part of the vulgar to suspect poisoning. They never can be brought to consider, that persons may die a natural death suddenly, as well as slowly; or, as we shall presently see, that death may really take place slowly, as in cases of disease, and yet be due to poison. This prejudice continually gives rise to the most unjust suspicions of poisoning; a case illustrating this has already been given (ante, p. 112). One of the means recommended for distinguishing narcotic poisoning from apoplexy, or disease of the heart, is the difference of the rapidity with which death takes place. Thus, apoplexy, or disease of the heart, may prove fatal either instantly or within an hour. The only common poison likely to operate with such fatal rapidity is prussic acid. But when this is the cause of death, some traces of the poison may be found at hand, except in a case of murder, when it has been intentionally removed. Poisoning by opium is commonly protracted for five or six hours. This poison has never been known to destroy life instantaneously, or in a few minutes. Thus, then, it may happen that death will occur with such rapidity, as to render it impossible to attribute it to narcotic poison under the circumstances.

The following case is reported by Anglada: A lady, in perfect health, while supping with her husband and family, complained, after having taken two or three mouthfuls, of severe pain in the region of the heart. She fell back in her chair, and died instantly. The parties not having lived on the best terms, the husband was openly accused of having been accessory to the poisoning of his wife,—a circumstance which was rendered still more probable in the opinion of his neighbors, by the fact that his wife had lately made a holographic will in his favor. One of his servants, with whom he was said to live in adultery, was arrested, and a paper, containing a white powder, was found in her possession. The husband endeavored to compromise the affair by offering to give up the will. Here, then, were strong moral presumptions of death from poisoning. Three surgeons (experts!) were appointed to examine the body. They opened the abdomen, and observing some green spots in the stomach (produced, as it afterwards appeared, by imbibition from the gall-bladder), pronounced an opinion that the organ was in a gangrenous state from the effects of some corrosive poison. Some doubt arising on the correctness of this view, four other surgeons were directed to re-examine the body. They found that the stomach had not even been opened,—and that its mucous membrane, as well as that of the intestines, was perfectly healthy! It contained a small quantity of undigested food, which was free from any trace of poison. The deceased had died from natural causes. The white powder found in the possession of the servant was nothing more than white sugar. Had the usual effects been attended to by the parties, who were first called to give evidence in this case, it is obvious that no charge of poisoning could have been made with any shadow of probability. The deceased died suddenly: there is no common poison which acts so rapidly, except strong prussic acid in a very large dose. It was very unlikely that this should have been administered in solid food. Besides, there was no vomiting before death; hence it followed that, if poison were the cause, the whole of it must have been found in the viscera; but none was discovered. It is also to be observed, that any poison which destroys life within half an hour, or an hour, generally produces a train of well-marked symptoms, *i. e.* delirium, stupor, coma, convulsions, paralysis, or tetanus in a severe form.

The great utility of the coroner's inquest, in England, when the inquiry is properly conducted, is seen in its removing unfounded suspicions, which often arise in cases of sudden death.

Chronic Poisoning.—When a poison destroys rapidly, it is called a case of *acute* poisoning, to distinguish it from the *chronic* form, *i. e.* where death takes place slowly. Chronic poisoning is a subject which, of late years, in reference to arsenic, antimony, and lead, has required medico-legal investigation. Most poisons are capable, when their effects are not rapidly manifested, either from the smallness of the dose or from timely treatment, of slowly undermining the powers of life, and killing the patient by producing emaciation and exhaustion. This is sometimes observed in the action of arsenic and corrosive sublimate, but it has been remarked also, in cases of poisoning by the mineral acids and caustic alkalies. Death is here an indirect consequence: stricture of the œsophagus is induced, or the lining membrane of the stomach is destroyed, and the process of digestion impaired,—a condition which leads to innutrition and death. The time at which these indirect effects will prove fatal, is, of course, liable to vary. A person has been known to die from a stricture of the gullet brought on by sulphuric acid, *eleven months* after the poison had been swallowed; and there is no reason to doubt, that instances may occur of a still more protracted kind. In these cases of chronic poisoning, there is considerable difficulty in assigning death exclusively to the original action of the poison, since the habits of life of a person, a constitutional tendency to disease, and other circumstances, may have concurred to accelerate or produce a fatal result. To connect a stricture of the gullet with the act of poisoning by a mineral acid, it is necessary to show that there was no tendency to this disease before the acid was administered; that the symptoms appeared soon after the first effects of the poison went off; that these symptoms continued to be aggravated until the time of death; and that there was no other cause to which death could reasonably be referred. These remarks apply equally to the indirectly fatal effects of any poison, —such, for instance, as the salivation occasionally induced by corrosive sublimate, when the acute symptoms of poisoning by that substance have passed away, and to that form of paralysis which is supposed to have been induced by chronic lead-poisoning. Several cases of criminal poisoning in a chronic form have come before our tribunals. Among others I may refer to those of Miss Blandy, tried, at Oxford, in 1752, for the murder of her father by arsenic; of a woman, named Butterfield, tried, at Croydon, in 1775, for the murder of a Mr. Scawen, by administering corrosive sublimate; to the case of Wooler (Durham Winter Assizes, 1855), in which the use of arsenic had extended over a period of about seven weeks; of Ann Palmer (January, 1856), who died from repeated doses of tartar emetic administered to her by William Palmer; and the case of McMullen (Liverpool Autumn Assizes, 1856), in which death was caused by small doses of tartar emetic frequently repeated. In most cases, criminals destroy life by administering poison in large doses; but, in these instances, small doses were given at intervals,—a fact which led to a medical doubt of the real cause of the symptoms before death.

It has been already remarked, that some poisons have what is called an *accumulative* property, *i. e.* they may be administered for some time in small doses without producing any marked effects; but they will, perhaps, after a certain period, suddenly and unexpectedly give rise to violent symptoms affecting the life of a person. This peculiar mode of action has been witnessed more in medical practice than in cases of attempts to poison; hence it is not a subject of much importance to a medical jurist. Foxglove (digitalis) is said to possess this property; it has been remarked, on more than one occasion, that persons to whom this medicine had been repeatedly administered in small doses have died suddenly, probably from the accumulative properties of the poison. The same effect has been noticed in the use of chloroform vapor, and in the case of other poisons.

This accumulative property is probably dependent on the retention of the poison in the blood. It is not eliminated by the lungs or excretions with sufficient rapidity, and it is not deposited in the organs. The point of saturation of the blood varies, it is believed, with each poison, and ranges within small limits. The rate of elimination is also known to be variable. These facts may serve to explain why it is that the accumulative property is manifested. The dose may not have been increased, but the blood may still retain so much of the poison that a very small addition may render it fatal. In the operation of some poisons through the lungs, *e. g.*, the diluted vapor of chloroform, it has been found that dangerous consequences may ensue if a maximum effect is produced under the continued respiration. It has been found better to withdraw the vapor before the full effect is produced, as the insensibility increases after the removal of the vapor. The full effect of the poison on the organic and nervous system is not at once induced.

2. EVIDENCE FROM APPEARANCES AFTER DEATH.—One of the principal means of determining whether a person has died from poison, is an examination of the dead body. In relation to *external appearances*, there are none indicative of poisoning upon which we can safely rely. It was formerly supposed, that the bodies of persons who were poisoned, putrefied more readily than those of others who had died from natural disease; and evidence for or against poisoning was at one time drawn from the external appearance. This is now known to be an error; the bodies of persons poisoned are not more rapidly decomposed, *cæteris paribus*, than those of others who have died a sudden and violent death from any cause whatever.

Irritant poisons act chiefly upon the stomach and intestines, which they irritate, inflame, and corrode. We may likewise meet with all the consequences of inflammation, such as ulceration, perforation, and gangrene. Sometimes the coats of the viscera are thickened, at other times thinned and softened by the action of an irritant.

Among *Neurotic* poisons—those which have a cerebral action, and the true spinal poisons (strychnia and brucia), do not commonly leave any well-marked appearances. The stomach and intestines present no unnatural changes. There is greater or less fulness of the bloodvessels of the brain or its membranes; but even this is often so slight as to escape notice, unless attention is particularly directed to the brain. An effusion of blood is rarely found. Some of the neurotic poisons which affect both the brain and spinal marrow (aconite) may produce congestion of the brain or of its membranes, as well as marks of irritation in the stomach and bowels, and commonly both, according to their peculiar mode of action.

The *Spinal* poisons, in the form of alkaloids, present no well-marked or uniform appearances in the dead body.

It is important to bear in mind, that both irritant and neurotic poisons may destroy life without leaving any appreciable changes in the body. To such cases as these, the remarks about to be made do not apply. The proofs of poisoning must, then, be obtained from other sources. Any evidence derivable from appearances in the body of a person poisoned, will be imperfect unless we are able to distinguish them from those analogous changes, often met with as the results of ordinary disease. These are confined to the mucous membrane of the stomach and bowels. They are redness, ulceration, softening, and perforation. Any of these conditions may depend upon disease, as well as upon the action of irritant poisons.

REDNESS.—It is a main character of the irritants to produce redness of the mucous membrane of the stomach and small intestines. This redness, when first seen, is usually of a deep crimson color, becoming brighter by exposure to air. It may be diffused over the whole mucous membrane: at other times it is seen in patches, dots, or streaks (striæ), over the surface of the stomach. It is sometimes met with at the smaller, but more commonly at the larger end of this organ; and again, we occasionally find that the folds or prominences only of the mucous membrane present this red or inflamed appearance.

Redness of the mucous membrane may, however, be due to gastritis or gastro-enteritis (ante, p. 125); and in order to assign the true cause, it will be necessary to have an account of the symptoms preceding death, or some proof of the existence of irritant poison in the contents of the stomach or the tissues of the body. In this respect the case (Reg. v. Hunter, Liverpool Spring Assizes, 1843), is of some importance. A woman was charged with having poisoned her husband by arsenic. The medical evidence rested chiefly on the symptoms and appearances after death, for no arsenic was discovered in the body. The mucous membrane of the stomach and intestines was found, throughout its whole extent, much inflamed and softened. The medical witnesses for the prosecution referred this condition to the action of arsenic; those for the defence considered that it might be owing to idiopathic (spontaneous) gastro-enteritis, independently of the exhibition of any irritant substance. The circumstances of the case were very suspicious: but the prisoner was acquitted, not merely on account of the variance in the medical evidence, but from the absence of positive proof of the presence of poison in the body, *i. e.* its detection by chemical analysis (see ante, p. 49).—(See the published reports of the case by Mr. Holland and Mr. Dyson.)

In the healthy state, the mucous membrane of the stomach is pale and white, or nearly so, except during digestion, when it becomes reddened; and some observers have remarked that a slight redness has often remained in the stomachs of those who have died during the performance of the digestive process. When in contact with the spleen or liver in the dead body, the stomach is apt to acquire a deep livid color from the transudation of blood; and it is well known that the bowels acquire a somewhat similar color from the gravitation of blood, which always takes place after death. None of these appearances are likely to be mistaken for the action of an irritant poison.

There is an important class of cases in which redness of the mucous membrane of the stomach is found after death, and is not dependent on the action of poison or on any assignable cause. These cases, owing to their being so little known, and involved in much obscurity, deserve great attention from a medical jurist; since the appearances closely resemble those produced by irritant poison. A person may die without suffering from any symptoms of disordered stomach; but, on an inspection of the body, a general redness of the mucous membrane of this organ will be found, not distinguishable from the redness which is so commonly seen in arsenical poisoning. Several cases of this kind have occurred at Guy's Hospital; and drawings have been made of the appearances of the stomach. A record has been kept of four of these; and it is remarkable that, although in not one of them, before death, were any symptoms observed indicative of irritation or disease of the stomach, yet in all, the stomach was found more or less reddened, and in two extensively so. Such cases are not very common; but the certainty of their having occurred where poisoning could not be suspected, should place the witness on his guard, so that he may not be led to countenance a suspicion of poisoning too hastily. In order to distinguish them, we must note whether there have been any symptoms during life, and their nature; as in the above cases, there may have been no symptoms, or they may have amounted only to slight disturbance of the stomach. Under these circumstances, they could not be mistaken for symptoms of irritant poisoning. Such cases are only likely to lead into error those who trust to this appearance alone as evidence of poisoning; but no medical jurist, aware of his duty, could ever be so misled.

This pseudo-morbid redness of the stomach may truly occur where there is some ground for suspicion, as in the following case, communicated to the Medical Gazette by Mr. Tyson, of Beccles. A young woman, far advanced in pregnancy, died suddenly in a fainting fit, one morning soon after she had risen. She had been in ill health previously; but there was nothing to indicate that she had taken poison; indeed, from what has been already said, the suddenness of her death was rather adverse to the suspicion that she had died from such a cause. Yet after death it was found, among other appearances, that the mucous membrane of the stomach was inflamed (reddened?) and raised in folds. There had been no symptoms of irritation in the stomach or bowels. (A case, in which it is probable this pseudo-morbid appearance of the mucous membrane was mistaken for the effects of irritant poison, will be found in the Ann. d'Hyg. 1835, i, 227.) Dr. Yelloly long since remarked, that the mucous membrane of the stomach often presented a high degree of vascularity (redness) in cases of sudden death. He met with this appearance in the stomachs of some executed criminals, whose bodies were examined soon after they had undergone the sentence of the law. From his observations it appears—1. That vascular fulness of the lining membrane of the stomach, whether florid or dark-colored, is not a special mark of disease, because it is not inconsistent with a previous state of perfect health. 2. That those pathologists were deceived, who supposed, from the existence of this redness in the stomach, that gastritis sometimes occurred without symptoms. 3. That erroneous conclusions on the cause of death were frequently owing to the same mistaken observations: the effects of putrefaction and spontaneous changes, induced by the loss of vitality, being sometimes attributed to the action of poisons. 4. That the redness in question is entirely venous, the florid state of the vessels arising from the arterial character of the blood remaining in the veins for some time after its transmission from the arterial capillaries at the close of life: the appearance is, however, sometimes due to transudation only. 5. That the fact of inflammation having existed previously to death, cannot be inferred merely from the aspect of the vessels in a dead part: there must at least have been symptoms during life. (See Medical Gazette, vol. xvii, p. 309.) Andral and other pathologists have adopted similar views, and these views have obviously an important bearing upon medico-legal practice: since there is generally a tendency to suspect poisoning, whenever redness of the mucous membrane of the stomach is met with in the dead. Such a condition does not even prove the past existence of inflammation, unless there were symptoms during life or other marked effects of the inflammatory process in the alimentary canal. It can be no sign of poisoning, unless the presumption is supported by evidence from symptoms, or by the actual discovery of poison in the stomach or other viscera.

In the case of Good, executed some years since, the mucous membrane of the stomach was found reddened, as if from the action of an irritant poison; and, in one instance of death from asphyxia by carbonic acid, there was not only reddening but effusion of blood in the stomach. The deceased was found dead in her bed, and the husband was suspected of having destroyed her by poison. A minute investigation of the case showed that the suspicion was unfounded.

A case is mentioned by Foderé, where, in the body of a person who died suddenly, and poisoning was suspected, the gullet and stomach were found reddened. This was at first considered to be a confirmation of a suspicion, that the deceased had died from poison; but on inquiry, it was ascertained, that the redness was due to the coloring matter of a strong infusion of red poppies, which the deceased had been in the habit of taking. It is not likely that any person, moderately informed in his profession, would fall into such a mistake as this,—the means of distinguishing all red colors by chemical tests being so simple; not to mention that mere washing would generally remove colors of this kind.

The redness of the stomach, in cases of poisoning, is so speedily altered by putrefaction, when circumstances are favorable to this process, as to render it difficult for a witness to speak with any certainty upon its cause. Putrefactive infiltration from the blood contained in the adjacent viscera and muscles, will give a reddish-colored appearance to a stomach otherwise in a healthy condition. Great dispute has arisen respecting the length of time during which redness of the stomach produced by an irritant will be recognizable and easily distinguishable from putrefactive changes. It is, perhaps, sufficient to say, that no certain rule can be laid down on this subject: it must be left to the knowledge and discretion of the witness. It will depend on the nature of the poison taken and the degree of putrefaction. I have distinctly seen the well-marked appearances of inflammation produced by arsenic in the stomach and duodenum, in an exhumed body, twenty-eight days after interment (Reg. v. Jennings, Berks Lent Ass. 1845); and in another instance (August, 1846), the reddened state of the mucous membrane, in a case of arsenical poisoning, was plainly perceptible, on removing a layer of arsenic, *nineteen months* after interment. In the case of Mrs. Bacon, the redness of the stomach and bowels was apparent, although the body had been *twenty-one months* in the grave. (Reg. v. Bacon, Lincoln Summer Ass. 1857.) (See, on this question, a case of suspected poisoning, by Orfila, Annales d'Hyg. 1839, i, 127.) If, however, there be the least doubt respecting the origin of the redness, it would be proper not to rely upon it, as evidence of poisoning.

ULCERATION.—In irritant poisoning, the stomach is occasionally found ulcerated; this is, comparatively speaking, a rare occurrence in acute cases, but not unfrequent in chronic or protracted cases of poisoning by arsenic. In these cases, the mucous membrane is removed in small distinct circular patches, under the edges of which the poison (arsenic) is often found lodged. Ulceration of the stomach is perhaps a more common result of disease than of the action of poison. As a consequence of disease, it is very insidious, going on often for weeks together, without giving any indication of its existence, except perhaps slight disturbance of the stomach, with occasional nausea, vomiting, and loss of appetite. In this case the ulceration is commonly seen in small circumscribed patches. It is worthy of remark, as one means of distinction, that ulceration has never been known to take place from arsenic or any irritant poison, until *after* symptoms indicative of irritant poisoning have occurred. In ulceration from disease, the mucous membrane is commonly only reddened in the neighborhood of the ulcer. In ulceration from poison, the redness is generally diffused over other parts of the stomach, as well as over the duodenum and small intestines. A case, however, occurred in Guy's Hospital, some years ago, in which, with a small circular patch of ulceration near the cardiac opening, the whole mucous membrane was red and injected: but this singular condition of the stomach, so closely resembling the effects of an irritant poison, was unaccompanied by any marked symptoms during life. The history of a case previous to death, will thus commonly enable us to determine, to what cause the ulceration found, may be due. Care must be taken to distinguish ulceration from corrosion. Ulceration is a vital process; the substance of a part is removed by the absorbents as a simple result of inflammation. Corrosion, on the other hand, is a chemical action;—the parts are removed by the immediate contact of the poison: they are decomposed: their vitality is destroyed, and they combine with the corrosive matter itself. Ulceration requires time for its establishment, while corrosion is generally an instantaneous effect.

There is no form of acute poisoning, in which a medical jurist has it more in his power to pronounce an opinion from appearances in the dead body, than in cases of death from the mineral acids. Tartra long since observed, that whenever the alimentary canal, from the mouth to the intestines, was found corroded and converted to a soft fatty substance of a bright yellow or brown color, when it was easily detached from the subjacent parts, and there were marks of inflammation or gangrene or actual perforation of the stomach, there could be no doubt that these effects were due to a mineral acid, whether the acid were discovered in the body or not. Dr. Christison has also adopted this view; and he very properly remarks that such cases must be considered as distinct exceptions to the general rule, regarding the weakness of evidence derived from appearances after death. Indeed, it may be inquired of those who are disposed to entertain an adverse opinion, what conceivable form of disease can produce such well-marked appearances simultaneously in the mouth, throat, gullet, stomach, and intestines, in the course of a few hours.

SOFTENING.—The coats of the stomach are not unfrequently found so soft, as to yield and break down under very slight pressure; and this may be the result either of poisoning, of some spontaneous morbid change in its structure during life, or of the solvent action of the gastric juice after death. As this change in the stomach, when caused by poison, is commonly produced by those substances only which possess corrosive properties, it follows that in such cases, traces of their action will be perceived in the mouth, throat, and gullet. In softening from disease, the change will be confined to the stomach alone, and it is commonly found only at the cardiac extremity of the organ. When softening is really caused by an irritant poison, it is generally attended by other striking and unambiguous marks of its operation. Softening is not to be regarded as a common character of poisoning; it is only an occasional appearance. In one case of arsenical poisoning, the coats of the stomach were thickened almost of a gelatinous consistency, and free from any redness as a result of inflammation. I have met with a case, in which the coats of the stomach were considerably hardened by sulphuric acid, and in one instance, hardened and thickened by arsenic. Softening can never be inferred to have proceeded from poison, unless other well-marked changes are present, or unless the poison be discovered in the softened parts. The stomachs of infants have been frequently found softened from natural causes;—such cases could not be mistaken for poisoning, since the history during life,—the absence of symptoms and of other appearances indicative of poisoning, as well as the total absence of poison from the viscera, would prevent such a suspicion from being entertained.

PERFORATION.—The stomach may become perforated either as a result of poisoning or disease. 1. *Perforation from poisoning.*—This may occur: 1. By corrosion; 2. By ulceration. The perforation by *corrosion* is by far the most common variety of perforation from poisoning. It is occasionally witnessed when the strong mineral acids have been taken, especially sulphuric acid: the stomach, in such cases, is blackened and extensively destroyed,—the aperture is large, the edges are rough and irregular, and the coats become easily lacerated. The poison escapes into the abdomen, and may be readily detected by chemical analysis. The perforation from *ulceration*, caused by irritant poison (arsenic), is but little known. There are, so far as I know, but few instances on record. In a great number of poisoned subjects examined during many years past at Guy's Hospital, not a single case has occurred. It must then be looked upon as a rare appearance in cases of irritant poisoning. In fact, the person dies from the specific effects of the irritant poison before there is time for perforation by ulceration to take place.

2. *Perforation from disease.*—This is by no means an unusual occurrence. Many cases of this description will be found reported elsewhere. (Guy's Hosp. Rep. No. 8.) It is invariably fatal when it proceeds so far that the contents of the stomach escape into the abdomen; but sometimes the stomach adheres to the pancreas during the ulcerative process, and then the person may recover. Several specimens of this kind of adhesion have been met with in inspections. The symptoms from perforation commonly attack a person suddenly, apparently while enjoying perfect health. Thus, then, these cases may be easily mistaken for those of irritant poisoning. The principal facts observed with regard to this formidable disease are the following: 1. It often attacks young females from eighteen to twenty-three years of age. 2. The preceding illness is extremely slight, sometimes there is merely a loss of appetite or capricious appetite, with uneasiness after eating. 3. The attack commences with a sudden and most severe pain in the abdomen, generally soon after a meal. In irritant poisoning, the pain usually comes on gradually, and slowly increases in severity. 4. Vomiting, if it exist at all, is commonly slight, and is chiefly confined to what is swallowed. There is no purging: the bowels are generally constipated. In irritant poisoning, the vomiting is usually severe, and purging seldom absent. 5. The person dies commonly in from eighteen to thirty-six hours;—this is also the average period of death in the most common form of irritant poisoning, *i. e.* by arsenic; but in no case yet recorded, has arsenic caused perforation of the stomach, within twenty-four hours; and it appears probable that a considerable time must elapse before such an effect could be produced by this or any irritant. 6. In perforation from disease, the symptoms and death are clearly referable to peritonitis. 7. In the perforation from disease, the aperture is commonly of an oval or rounded form, about half an inch in diameter, situated in or near the lesser curvature of the stomach, and the edges are smooth. The outer margin of the aperture is often blackened, and the aperture itself is funnel-shaped from within outwards, *i. e.* the mucous coat is the most removed, and the outer or peritoneal coat, the least. The coats of the stomach, round the edge of the aperture, are usually thickened for some distance; and when cut, they have almost a cartilaginous hardness. These characters of the aperture will not alone indicate, whether it be the result of poisoning or disease; but the absence of poison from the stomach, with the want of other characteristic marks of irritant poisoning, would enable us to say, that disease was the cause. Besides, the history of the case during life, would materially assist us in our judgment. The great risk in all these cases is, that the effects of disease may be mistaken for those of poison-

ing; for we are not likely to mistake a perforation caused by irritant poison for the result of disease.

Among numerous instances, tending to show the medico-legal bearings of this subject, I shall select one, which came before Mr. Hilton and myself for examination. A female in a noble family, aged twenty-three, died somewhat suddenly, under suspicious circumstances. She had been unwell for about three weeks, and was subject to occasional vomiting and disorder of the stomach. Still, her illness was so slight that it did not in the least interfere with the performance of her usual duties. One afternoon, about four o'clock, and about three hours after her last meal, she was suddenly seized with the most excruciating pain in the abdomen, and violent vomiting. Her skin was cold and clammy, and the abdomen tender and painful. It was suspected that she had taken poison; and magnesia and sulphate of magnesia were given to her. No poison was found in the room, and she strongly denied the imputation. The symptoms became worse, the vomiting more violent, and she died the following morning, about fifteen hours after her first seizure. On inspection, all the organs were found healthy, except those of the abdomen. There were here strong marks of peritoneal inflammation; the intestines were loosely adherent to each other, and a quantity of lymph was effused around them. The cavity contained about a pint of liquid, which had escaped from an aperture in the stomach. This liquid was reserved for analysis. The stomach was laid open by making an incision along its greater curvature. It was empty. At the upper and posterior part, near the pyloric end of the smaller curvature, was an opening of an oval shape, about half an inch in its longest diameter. The edges were firm, hard, and smooth, presenting not the least appearance of laceration or ulceration. They were bevelled off from within outwards, being thinned towards the peritoneal coat, the aperture in which was much smaller than in the mucous membrane. There was no sign of inflammation in the membranes around; but the peritoneum, about the edge of the aperture, had a black appearance, and the coats of the stomach were thickened. At the lower part, near the larger curvature, there were thick, irregular lines of blackness (striæ), the mucous membrane being raised and blackened, but not softened. These black lines appeared like those produced by sulphuric acid; but there was no corrosion, and on applying test-paper there was no acid reaction. The black matter was interspersed with a yellowish-colored substance. The liquids taken from the abdomen, as well as the coats of the stomach, were chemically examined; but not a trace of poison could be detected. Considering the time of the occurrence of symptoms, their nature, and the absence of poison from the viscera and their contents,—the suspicion of poisoning was at once negatived, especially when the above facts were taken together with the appearances in the dead body. The medical opinion given was, 1. That the deceased had died from peritonitis, caused by extravasation of the contents of the stomach. 2. That this extravasation was owing to a perforation of the coats of the organ, caused by the slow and insidious disease, and not by poison. (See Guy's Hospital Reports, October, 1850, p. 226.) In another suspected case the body was exhumed after several months' burial, and on examination, the stomach was found perforated in the usual situation. There was no poison.

It has been hitherto supposed that perforation of the stomach must necessarily prove fatal. This is undoubtedly the ordinary result, but the fatal effect depends on peritonitis, excited by effusion of the contents of the organ. Under favorable circumstances, and by judicious treatment, no effusion may take place, and the person may entirely recover; as the aperture is ultimately closed by adhesion to the surrounding viscera. Of this closure of ulcerated apertures in the stomach, several specimens are preserved in the museum of Guy's Hospital. This fact is of importance to the medical jurist, as the case might, from the symptoms, be mistaken for one of poisoning; although, when taken on the whole, they are unlike those produced by irritant poison. An apparently well-marked instance of recovery from perforation is reported by Dr. Hughes. (G. H. Rep. N. S. iv, 332.) The patient recovered from the first attack, but ultimately died from another perforating ulcer, which led to extravasation. (Case by Mr. Hilton, ib. 343.)

Spontaneous or Gelatinized Perforation.—The stomach is occasionally subject to a spontaneous change, by which its coats become softened and give way, generally at the larger end. As the effusion of the contents of the organ in such a case never gives rise to peritoneal inflammation, and no symptoms occur prior to death to indicate the existence of so extensive a destruction of parts, it is presumed that the stomach undergoes a process of solution soon after death. It is commonly attributed to the solvent action of the gastric juice,—the spleen, diaphragm, and other viscera in contact with the stomach being sometimes softened. (For some remarks on this subject, by Dr. Budd, see Med. Gaz. xxxix, 895.) In January, 1845, I met with an instance of this perforation in a child between two or three years of age. It was seized with convulsions, became insensible, and died in twenty-three hours. After death, the cardiac end of the stomach was found destroyed to the extent of three inches; and the edges were softened and blackened. There was no food in the stomach, nor had anything passed into the organ for thirty-two hours before death! It was therefore impossible to ascribe death to the perforation, or the perforation to poison. (For a full account of this case, see Med. Gaz. xxxvi, 32.) In October, 1846, I found a similar condition of the stomach in an infant aged nineteen months, suspected to have died from poison administered to it three months before. The cause of death in this case was mesenteric disease. The stomachs of children at the larger end are always very thin, and thus but a slight softening action is required to bring about the destruction of the coats. The same effect is observed in rabbits, in which the coats of the stomach are remarkably thin, and are often found quite pulpy on inspection, irrespective of disease or the presence of poison.

This form of perforation is reported to have been met with in the bodies of children affected with water on the brain,—in those who have died from typhus fever;—and, according to Andral, in females who have died during parturition. Dr. Macintyre informed me that he had met with two cases of this kind of perforation in young subjects affected with diabetes. The conditions for its production, whether local or constitutional, and the circumstances under which it occurs, are very obscure. (Med. Gaz. xxxix, 897; also vol. xli, p. 293.) The fact of most importance to the medical jurist is, however, that it is unattended by any marked symptoms during life. Some French pathologists describe cases of what they term *gelatinized* perforation, in which disorder of the stomach had existed. Chaussier, indeed, believed that this form of perforation always depended on a particular disease of the organ; and he denied, from the results of his own experiments, that the gastric juice had any solvent action. (Flandin, Traité des Poisons, i, 259.) The inspection of the body, with a general history of the case, must, however, suffice to remove any difficulty in forming an opinion whether the extensive destruction commonly met with, has or has not arisen from poison. Thus, in a cadaveric perforation, the aperture, which is always situated in that part of the stomach which lies to the left of the opening of the gullet, is very large, of an irregular form, and ragged and pulpy at the edges. These have the appearances of being scraped; the mucous membrane of the stomach is not found inflamed. There is occasionally slight redness, with dark brown or almost black lines or streaks (striæ), in and near the dissolved coats, which have an acid reaction. It can only be confounded with perforation by the action of corrosives; but the well-marked symptoms during life, and the detection of the poison after death, together with the changes in the throat and gullet, will at once indicate the perforation produced by corrosive poison. The only case in which any mistake is likely to occur, is where, conjoined with the discovery of perforation after death, there may have existed some symptoms of irritation in the stomach and bowels during life. It is possible that a person may die under symptoms somewhat resembling irritant poisoning, and after death the gastric secretion may destroy the coats of the stomach; but such a singular combination of circumstances must be most unusual. This, however, signifies little in a legal point of view, for persons charged with the crime of poisoning, are frequently acquitted on the barest medical possibilities. One case of this doubtful character is on record. I allude to that of Miss Burns, for the murder of whom, by poison, a Mr. Angus, of Liverpool, was tried in the year 1808. It is not necessary to enter into the particulars of the case; since the appearances in the body are imperfectly described in the report. Although the symptoms, resembling irritant poisoning, under which the deceased labored, were not accounted for, yet there was great reason to believe that they were not connected with the perforation of the stomach, which, on the whole, bore the characters assigned to that produced by the gastric secretion. The charge of poisoning was not sustained either by the chemical or the pathological evidence, and the prisoner was acquitted. The evidence given on this trial is well worthy of the attention of every medical practitioner. It shows on what a nice balance of proofs charges of poisoning sometimes rest, and how important it is that a medical jurist should make himself acquainted with all the circumstances under which perforations of the stomach may occur.

Perforations of the Œsophagus (*gullet*) *and Intestines.*—Other parts of the alimentary canal are liable to become perforated; but not, so far as I have been able to ascertain, by the action of irritant poison. The gullet may become softened and destroyed by the contact of corrosive poison, but, in general, this rapidly passes through the tube and lodges in the stomach. There is one instance on record in which the gullet was completely destroyed by the action of sulphuric acid. As Dr. Christison observes, it is not probable that a corrosive poison could ever perforate the intestines from within outwards, since its action would be chiefly expended on the stomach, and it is not likely to reach any portion of the intestines in a state sufficiently concentrated to destroy their coats by chemical action. (On Poisons, 149.) If a large quantity of corrosive poison flowed through an aperture in the stomach upon the intestines, then the coats might become destroyed from without inwards. The lower portion of the œsophagus, and various parts of the intestinal tube, have been found in several instances softened and destroyed, the aperture presenting all those characters which have been described in speaking of spontaneous or gelatinized perforation of the stomach. This change in the œsophagus is ascribed to a solvent action of the gastric juice, which enters the tube by regurgitation; but this explanation cannot apply to the intestines. A case of softening of the intestines, from the duodenum to the sigmoid flexure of the colon, is reported by Mr. J. Smith: the child died of water on the brain. (See Med. Gaz. ii, 619.) The intestinal tube may become perforated in any part by ulceration, either as a result of disease or of the action of irritant poison; but ulceration of the intestines from poison proceeding to a perforation of the coats, is a very rare condition.

Cases of *perforation of the intestines from disease* are occasionally met with. They require the attention of a medical jurist, since they may be easily mistaken for cases of poisoning. The following instance of perforation of the duodenum, is reported by Mr. Bailey (Med. Times, Dec. 19, 1846, p. 223). A female, æt. 28, was taken suddenly ill. There was great anxiety of countenance, small and frequent pulse, cold extremities, with vomiting and occasional eructations of wind. She suffered severe pain in the region of the small intestines (duodenum), which caused her body to be doubled up. Her bowels had been only partially opened the day before. In about nine hours there was great tenderness of the abdomen; the pulse was smaller; but the bowels, in spite of the administration of medicine, were still unmoved. She gradually sank, and died in about fourteen hours and a half from the time she was first seized. Thus far the symptoms would indicate that the case was one of acute peritonitis, and not of irritant poisoning. Nevertheless, the circumstances were extraordinary, and rumors were spread that her husband had poisoned her. The deceased, it appeared, had been married only the previous day: she and her husband did not retire to rest until three o'clock in the morning, and the attack came on suddenly, by a fit of severe pain, at nine o'clock, *i. e.* six hours after they had retired to rest. An inspection of the body showed all the usual marks of peritoneal inflammation, and the duodenum, in its transverse portion, was found to have a circular opening in it, surrounded by a black margin externally; while internally the perforation seemed larger, its sides sloping off. The mucous membrane was softened for some distance around the ulceration, affording evidence of the existence of previous inflammation. It appeared that the deceased had been a cook up to the day of her marriage. The only symptom manifested before the attack was, that she had been for some time subject to pain in her right side. (See also Cormack's Ed. Journ. June, 1845, p. 445; and Lancet, July 18, 1846, p. 67.)

A case of perforation of the small intestines (ileum), in which two apertures were found, is reported by Dr. Zartmann. (Caspar's Wochenschrift, März 14, 1846.) Death took place in two days, from inflammation of the peritoneum. One aperture was only the sixth of an inch in diameter. The edges of both were smooth, as if punched out.

Perforation by foreign bodies.—There is another insidious form in which perforation of the intestines may present itself, and cause fatal peritonitis. This is by the formation of an ulcerated aperture in the appendix of the cæcum (vermiformis cæci), of which two cases have been communicated to me by a former pupil, both occurring in young men. The perforation was produced in these instances by the pressure of a hard substance lodged in the extreme end. In one instance I analyzed this hard concretion, and found it to consist of inspissated mucus, biliary matter, and a large quantity of carbonate of lime. It was of an irregular form and structure, and about the size of a large pea. In both instances death was caused by peritonitis, produced by extravasation of the contents of the intestines, and the aperture was so small that it might have been easily overlooked. It is remarkable that the introduction into the appendix cæci of any foreign substance, as the pip of an apple, or a small bean, or cherry-stone, is liable to excite fatal inflammation, ulceration, or gangrene, and death. A case of this kind has been reported by Mr. Nelson (Amer. Jour. Med. Science, Feb. 1847, p. 258). The fact is important in relation to the causes of sudden death.

Perforation by worms.—It is now generally admitted that the various species of worms which infest the intestines of the human subject, may, in some rare cases, by irritating or even perforating the coats, give rise to symptoms which may be mistaken for those of poisoning. A recent writer on toxicology, M. Flandin, denies that perforation can ever take place from this cause (Des Poisons, i, 304, 1846); but as this denial is based on theory, while the statement which he impugns rests upon good authority, it is unnecessary to discuss this as an unsettled question. He supports his opinion by the authority of Rudolphi, who states that the entozoa have no organs capable of perforating the intestinal coats,—by the view entertained by M. Dujardin, that worms are never injurious to animals, because they are often found in large numbers when the previous health of the animal has been uninjured,—and by the statement of Andral, who, however, merely says, that if worms have the power of perforating the intestines, the cases in which this happens, are exceedingly rare—a conclusion in which most pathologists will entirely agree. Several instances are on record in which perforation has been thus caused, and the worm or worms have been found in the peritoneal cavity. A well-marked case of perforation of the small intestines (ileum) by an ascaris lumbricoides, leading to death from effusion and peritonitis, is reported by Mr. Kell. The opening through the intestine was of a circular form, and corresponded in magnitude with the size of the worm, which was eight inches long. The worm was found among the intestines, between the umbilicus and pubes. (Med. Gaz. ii, 650.) These worms may even perforate the abdominal parietes. (See cases by Dr. Young, Med. Gaz. ii, 748.) Vogel says of the ascaris lumbricoides, that in certain cases it is capable of perforating the intestine by thrusting asunder with its head the fibres of the intestinal coats. (Pathological Anatomy, Day's translation, p. 464.) When perforation takes place from this cause, it will be indicated by the discovery of the entozoon in the cavity of the peritoneum, or near the aperture in the intestine. (See a case in Cormack's Ed. Journ. June, 1845, p. 447.)

In a case of suspected irritant poisoning, when worms are discovered in the intestinal canal, an attempt is not unfrequently made in the defence to refer the symptoms of irritation to the presence of these entozoa. If the symptoms of poisoning be well marked, and some of the poison be discovered in the body, such a defence must be a hopeless struggle against medical facts. This objection was taken to the evidence from symptoms in one case of arsenical poisoning; but the disturbance occasioned by worms is commonly slight, and is very rarely accompanied by vomiting and purging. A solitary instance is quoted by Dr. Christison, in which a child appears to have died under symptoms of narcotico-irritant poisoning as an effect of worms. Several hundred ascarides were found in the intestines, and thirteen in the stomach. (On Poisons, 183.) In two instances of arsenical poisoning, I found a large ascaris lumbricoides in the small intestines, and in one instance recently (April, 1858), three of these worms, from six to eight inches long, were found living in the duodenum a week after the death of the person, who had been poisoned. In the above cases there could not be the slightest doubt that the sole cause of death was poison. In one instance the ascaris was well washed from adhering mucus, and examined for arsenic by Reinsch's process: the poison was abundantly discovered in it. (G. H. Rep. N. S. iv, 462.) In the third case, the man had taken calomel shortly before death, and mercury was extracted from the body of the worm after it had been washed. Under such circumstances it would be impossible to refer the symptoms of irritation to worms. A case will be mentioned in a future chapter, in which, on examining the body of a person who had taken an ounce and a half of muriatic acid, the jejunum was found perforated by a lumbricus; but the acid was beyond all doubt the cause of death.

That, however, the presence of worms in the body may be made a ground of defence, is sufficiently proved by two cases reported by M. Flandin. A young man was charged with having poisoned his father with arsenic. A very small quantity of the (absorbed) poison was detected in the tissues of the body only. The report of the inspection stated, that there was a large number of the ascarides lumbricoides in the intestinal canal, and some were even found in the œsophagus. A discussion on the cause of death arose between the counsel employed in the defence, who based his arguments on a memoir by M. Raspail, and the medical witness, who had discovered arsenic in the body, but especially in the liver. It was contended that the quantity of arsenic found was not sufficient to cause death (for the fallacy of an argument of this kind, see post, Chemical Analysis); and that the presence of the ascarides explained satisfactorily the cause of the symptoms, and their rapidly fatal course. The deceased, it was alleged, had been suffocated by the worms, which had caused compression of the bowels, and had even ascended into the œsophagus! The witnesses replied, that arsenic even in small quantity was never found in the healthy human body,—that the presence of worms could not account for the presence of absorbed arsenic in the tissues,—while the detection of the poison, on the other hand, accounted for all the symptoms, even for the passage of the worms into the œsophagus as a result of violent vomiting. To no other circumstance could death be attributed.

In the second case, an empiric applied to the cancerous breast of a female, a plaster covered with arsenious acid. Symptoms of poisoning by absorption followed, and the woman died. Arsenic was detected in the liver. An ascaris was found in the intestines. On removing the whole of the viscera, which were in a highly putrefied state, a portion gave way, and the worm appeared through the lacerated aperture. There had been no extravasation, nor any mark of peritonitis. The defence was, that the deceased had died from perforation of the intestine by the worm. This was denied by the witnesses, who contended that, had this been the case, there would have been extravasation. The escape of the worm was owing to accidental laceration of the viscera during the inspection. The symptoms of the disease were clearly those of poisoning and not of peritonitis. (Des Poisons, i, 307 and 507.)

Five species of parasitic worms have been hitherto found in the human intestines: 1. *Ascaris lumbricoides*, or round worm. 2. *Trichocephalus dispar*, or long thread worm. 3. *Oxyuris*, or *Ascaris Vermicularis*, common thread worm. 4. *Tœnia solium*, common tape worm; and, 5. *Tœnia lata* (*Bothriocephalus*), broad tape worm.

1. The *Ascaris lumbricoides* is very common. It is a round cylindrical worm pointed at both ends, especially at the anterior extremity; it varies in length from one inch to ten or even fifteen inches. It is usually of a whitish or brownish red color, and occasionally blood-red. A delicate furrow runs along the body on both sides. The worm occurs especially in children, and is often found in great numbers without any disturbance of health. When accumulated in large numbers, the ascarides are liable to excite intestinal irritation.

2. The *Trichocephalus dispar* is a thin and thread-like worm, varying in length from an inch and a half to two inches. It is usually white, sometimes slightly colored; it frequently occurs in the large intestines, especially in the cœcum. According to Vogel, it is found, gregarious or solitary, in nearly half the subjects examined; it is firmly adherent by its capillary head to the mucous membrane. Those persons, in whose bodies this worm has been found in very large numbers have not exhibited any symptoms of its presence.

3. *Oxyuris, Ascaris Vermicularis.*—This is a thin white worm, smaller than the trichocephalus; it occurs in the large intestines, particularly in the rectum, and it is especially common in infants. The male, which is smaller than the female, varies in length from a line to a line and a half.

It is spirally coiled at the tail, and often completely assumes the form of a ring.

4. *Tænia solium.*—A riband-shaped, long, pointed worm, of a milk-white or yellowish color. Its length sometimes exceeds twenty feet; its breadth varies from a quarter to half an inch, but it is less at the head; its greatest thickness does not exceed the twelfth of an inch. It inhabits the small intestines of the human subject, but only in certain parts of the world. It remains in the body for a long period, without its presence being indicated by the slightest symptom.

5. *Tænia lata.*—This worm closely resembles the preceding. It varies in length from one foot to twenty or even forty feet. There are but very slight differences between this and the tænia solium or vulgaris. (See, on this subject, Vogel's Pathological Anatomy, Day's translation, page 470.)

In investigating a suspected case of poisoning, when the person is not seen until after death, in addition to certain points elsewhere noticed in reference to the living (ante, p. 148), it will be proper that a medical jurist should direct his attention to those which follow. 1. The *exact time* of death, and thus determine how long a period the person has survived after having been first attacked with the symptoms. 2. Observe the attitude and position of the body. 3. Observe the state of the dress. 4. Observe all surrounding objects. Any bottles, paper-packets, weapons, or spilled liquids, lying about, should be collected and preserved. 5. Collect any vomited matters near the deceased. Observe, whether vomiting has taken place in the recumbent position or not. If the person have vomited in the erect or sitting position, the front of the dress will commonly be found covered with the vomited matters.

Inspection of the Body.—6. Note the external appearances of the body, and whether the surface be livid or pallid. 7. Note the state of the countenance. 8. Note all marks of violence on the person, or discomposure of the dress,—marks of blood, &c. 9. Observe the presence or absence of warmth, or coldness in the legs, arms, abdomen, mouth, or axillæ. 10. The presence of rigidity or cadaveric spasm in the body. To give any value to the two last-mentioned characters, it is necessary to observe the nature of the floor on which the body is lying, and whether this be clothed or naked, young or old, fat or emaciated. All these conditions create a difference, in respect to the cooling of the body and the access of rigidity. 11. If found dead—When was the deceased last seen living, or known to have been alive? 12. Note all circumstances leading to a suspicion of suicide or murder. 13. The time after death at which the inspection is made. 14. Observe the state of the abdominal viscera. If the stomach and intestines be found inflamed, the seat of inflammation should be exactly specified; also all marks of softening, ulceration, effusion of blood, corrosion, or perforation. The stomach should be removed and placed in a separate vessel, a ligature being applied at the cardiac and pyloric ends. 15. The contents of the stomach should be collected in a clean *graduated* vessel; notice, *a*, the quantity, *b*, the odor, tried by several persons, *c*, the color, *d*, acid or alkaline reaction, *e*, presence of blood, mucus, or bile, *f*, presence of undigested food; and here it may be as well to observe, that the presence of farinaceous matter (bread) would be indicated by the addition of iodine water, if the contents were not alkaline—of fat, by heat; *g*, other special characters. 16. The contents of the duodenum should be separately collected, ligatures being applied to it. 17. Observe the state of the large intestines, especially the rectum, as poison has been sometimes introduced into this portion of the bowels by injection, and note the condition of the contents. The discovery of hardened fæces would prove that purging had not existed recently before death. 18. The state of the mouth, larynx, throat, and gullet; whether there are in these parts any foreign substances or marks of inflammation and corrosion. This will throw light on the question, whether the poison swallowed was a local irritant or corrosive, and whether it had or had not a chemical action: 19. The state of the thoracic viscera; all morbid changes noted. 20. The state of the brain. 21. The condition of the genital organs should be examined, as in the female, poison has been sometimes introduced into the system by the vagina.

Such are the points to which, in the greater number of cases of suspected poisoning, a medical jurist should attend. By means of these data, as well as others (ante, p. 148), noted according to the particular case to which they are adapted, he will in general be enabled, without difficulty, to determine the probable time of death, the probable cause of death, and the actual means by which death was brought about. He may thereby have it in his power also to point out the dish which had contained the poison, if the case be one of poisoning; and to throw light upon any disputed question of suicide or murder in relation to the deceased. Many cases of poisoning are obscure, owing to these points not having been attended to in the first instance.

I have not considered it necessary to enter into any details respecting the mode of performing an inspection. This the practitioner will have acquired during his study of anatomy; and the only essential points in addition to those mentioned, are—1. To examine all the important organs for marks of natural disease; and 2. To note down any unusual pathological appearances, or abnormal deviations; although they may at the time appear to have no bearing on the question of poisoning. It is useful to bear in mind on these occasions, that the body is inspected, not merely to show that the individual has died from poison, but to prove that he has not died from any natural cause of disease. Medical practitioners commonly direct their attention exclusively to the first point; while lawyers, who defend accused parties, very properly direct a most searching examination to the last-mentioned point, *i. e.* the healthy or unhealthy state of those organs which are essential to life, and with which the poison has not probably come in contact. The most usual causes of sudden death commonly have their seat in the brain, the heart and its great vessels, and in the lungs. Marks of effusion of blood or serum, congestion, inflammation, suppuration, or a diseased condition of the valves of the heart, should be sought for and accurately noted, whatever may be the condition of the abdominal viscera, or of the parts which are specially affected by the poison.

CHAPTER XI.

ON THE EVIDENCE OF POISONING FROM CHEMICAL ANALYSIS—NOT ESSENTIAL—RULES FOR CONDUCTING AN ANALYSIS—CIRCUMSTANCES UNDER WHICH AN ANALYSIS MAY BE REQUIRED—FAILURE OF CHEMICAL EVIDENCE—CAUSES OF THE NON-DETECTION OF POISON—LOSS BY ELIMINATION AND PUTREFACTION—EVIDENCE FROM THE QUANTITY FOUND IN THE BODY—DELICACY OF ANALYSIS—POISONS IN TESTS AND APPARATUS—DANGER OF PREMATURE OPINIONS—FALLACIES IN CHEMICAL ANALYSIS.

Convictions without chemical evidence.—It has been supposed that chemical evidence of poisoning was always necessary, and that the *corpus delicti* was not made out, unless the poison were discovered by a chemical analysis. This, however, is not a correct view of the matter. There are many poisons which cannot, at present, be detected by chemical analysis, and among those susceptible of analysis there are numerous circumstances which, irrespective of a criminal tampering with the viscera, may occur to prevent their detection in the food, the vomited matters, or the contents of the stomach and bowels. (See the cases of Dr. Alexander (ante, p. 49); and Humphreys (ante, p. 113). If such a principle were recognized by law, many criminals would escape conviction. All that is required legally, is that there should be satisfactory proof of a person having died from poison;—the discovery of poison in the body is not necessarily evidence of its having caused death, nor is its non-discovery evidence that death has not been caused by it. If by the symptoms and appearances, with or without moral circumstances, it can be made clear to the minds of a jury that death has been caused by poison, nothing more is required; the evidence from chemical analysis may be then safely dispensed with. In cases of murder, the law commonly requires that the body of a deceased person should be produced, in order that the cause of death may be verified; but this is not absolutely necessary, for several convictions for murder have very properly taken place, where the bodies of the murdered persons have not been forthcoming. Thus, then, we must not suppose that a charge of poisoning cannot be sustained in the absence of chemical evidence of the nature of the substance taken. The fact of a poison having been used, as well as its nature, may be determined by physiological and pathological evidence, as well as by other circumstances. In the case of Donellan, already referred to, the only evidence of the nature of the poison used, was the odor perceived by a non-professional person. The effects which followed made up for the want of chemical proof of its nature. As some objections have been offered to the propriety of a conviction in this case, I may refer to others: one the case of a man named Thom, tried at the Aberdeen Autumn Circuit, 1821, for poisoning a person named Mitchell with arsenic. No trace of poison could be detected; but a conviction very properly took place on evidence from symptoms and appearances, coupled with moral circumstances. Another instance occurred at the Monaghan Lent Assizes, 1841, where a woman was convicted of poisoning her husband, although the nature of the poison could not be determined by the most carefully conducted chemical analysis. The poison was considered, from the alleged taste and symptoms, to have been aconite.

In Humphrey's case (ante, p. 113), the medical and moral facts rendered it clear that the man had died from the effects of sulphuric acid, administered to him by his wife; but not a trace of this poison could be detected in the body of the deceased, although he survived the effects only two days. The jury, however, were satisfied, from other proofs, that death had been caused by poison, and the wife, who committed the crime, was convicted and executed. In the case of Dr. Castaing, who was tried, in 1823, before a French tribunal, for the murder of his friend Ballet, by the administration of morphia, no trace of the poison was found in the body by several eminent chemists; yet the symptoms and moral evidence were considered to have furnished satisfactory proof of the crime, and the prisoner was convicted and executed.

Up to the period of the trial of William Palmer, for the murder of J. P. Cook (May, 1856), the facts established by the above cases had not been questioned by any person pretending to scientific knowledge. In this case no strychnia was found, although the medical and moral circumstances pointed clearly to death from this poison; and, taken as a whole, admitted of no other reasonable explanation. In spite of the most strenuous efforts to involve the question in the mysteries of pseudo-chemistry, the prisoner was convicted and executed.

It is now a well-known and admitted fact, that a person may die from poison, and no poison be found by chemical analysis in the body. There is a popular but erroneous notion, that, if poison cannot be produced from a dead body in a visible and tangible form, then, supposing proper skill to have been employed, the only inference to be drawn is, that no poison was taken, and that death was caused by disease. This would be bringing the question of death from poison to a very simple issue indeed. It would be casting aside physiology and pathology, and requiring our law-authorities to place entire and exclusive confidence in the crucible and test-tube of the chemist. But has organic chemistry, with all its modern advances, yet reached a point that no death can occur from poison, speaking generally, except the poison be still found either in the stomach, the tissues, the blood, the excretions, or in all of these parts at one and the same time? Is the poison of the viper or rattlesnake easily revealed by tests? Can the poison of rabies, producing one of the most formidable convulsive affections known, namely, hydrophobia, be detected in the tissues? Are there any chemical processes by which the poison of castor-oil-seeds,—of the common laburnum,—of the poisonous fungi,—of darnel, the sausage-poison of Germany, the poison of the œnanthe crocata, and the Woorara poison of South America,—can be separated and demonstrated to exist after death in the blood, liver, or tissues? If not, then the allegation that no person can die from poison, except the poison be found in the body, is a mockery, a delusion, and a snare, admirably adapted to cover a multitude of secret deaths from poison, which, but for this dogma, might be revealed by pathology and physiology. It is all the more dangerous, because the history of crime shows us that the arts of the murderer, especially of the scientific or professional murderer, are daily becoming more refined. I might add largely to the list of poisons which either by their nature, by their tremendous power in very small doses, or by the mode in which they are introduced into the system, would infallibly produce death without leaving a physical or chemical trace of their presence in the body. I forbear to do this. Such an enumeration would undoubtedly serve my purpose of refuting that which I believe to have been a gross and dangerous error on the part of some of the chemists who gave evidence for the defence at the trial of Palmer; but it would be at the cost of making public means of death and modes of perpetrating murder which it might be dangerous to promulgate. The fallacious doctrine here broached for the temporary purpose of saving the life of a wretched criminal was, however, such as to receive, at least for a time, a large amount of popular support. Alarm was also spread and allowed to pervade the public mind by the allegation, that unless poisons were invariably detected and separated, in cases of alleged poisoning, any innocent person might be convicted of murder by poison when death was really due to some latent disease. It is fortunate that the jury in Palmer's case have, by their verdict, given the deathblow to this novel and dangerous doctrine, and have shown that twelve men may be as safely directed to a just decision by the views of pathologists and physiologists as by the assumptions of chemists. This is as it should be. Chemistry may detect a poison; but it fails, without the aid of physiology and pathology, to show whether it was or was not the cause of death; and, in some instances, it cannot enable us to determine whether the poison was introduced into the body during life or after death. Even with regard to the poison in question in the case referred to,—*strychnia*,—this substance is now so extensively employed as a medicine, that the discovery of traces of it in the stomach, blood, and tissues (assuming that the processes used are satisfactory) would not justify an allegation of death from poisoning by it. *The symptoms must be made known.* The "tetanic complications" which it ordinarily produces in the body when taken in poisonous doses must be clearly established, and a judgment must be based on these symptoms. We are not, therefore, to suppose, as the public have been erroneously led to imagine, that toxicology and chemistry are convertible terms, that the finding of poison in a body is a proof of death from it, and the non-detection of poison is a proof of death from some natural cause. (See Guy's Hosp. Rep. Oct. 1856. Poisoning by Strychnia, p. 269.) William Palmer's case furnishes another illustration,—if any were needed,—that medical and moral, exclusive of chemical evidence, may suffice to procure conviction on a charge of poisoning.

It was alleged on the occasion of this trial, that, to teach the public that a person might die from poison and no poison be found in the body, was a doctrine most dangerous to the security of life. Whatever danger there may be in promulgating the facts, it is quite certain that a greater danger must attend the suppression of truth and the propagation of falsehood. The further experience of Mr. Herapath (a witness for the criminal Palmer) (see post, p. 190), has now enabled him to subscribe to the correctness of this doctrine, and, in one case at least, to swear that death had been caused by a poison of which, in spite of diligent search, he could find no trace in the body!

When the other branches of evidence are weak or defective, the detection of poison by chemical analysis is of such importance, that if it fail, an acquittal will follow. Conjoined with strong moral circumstances, chemical evidence will often lead to conviction when the appearances in the body are entirely wanting, and the evidence from symptoms is imperfect. The great value of chemical evidence, in otherwise doubtful cases of poisoning, is frequently shown in the detection of poison in bodies which are exhumed many months after burial, when all appearances are destroyed by decomposition. We cannot therefore be surprised to find that it is this branch of evidence which is deemed most satisfactory to the public mind, and which is earnestly sought for by our law authorities on charges of poisoning. The reason is, that in most cases, in the hands of a trustworthy analyst, it demonstrates at once the means of death; while symptoms and appearances, deposed to by inexperienced witnesses, are fallible criteria. Chemists, however, are not infallible, and instances might be adduced of their swearing in the most positive manner to the presence of poison in cases in which they have been afterwards obliged to admit that none existed! They have however, an advantage over the pathologists in the fact that the members of the medical profession generally are less competent to judge of the accuracy of a chemical, than of a pathological opinion. On the other hand it must be admitted, that some coroners are not sufficiently aware of the importance of the chemical branch of evidence in cases of suspicious death. In several instances, the fact of poisoning has been established by an analysis of the contents of the stomach long after interment, although verdicts of natural death had been previously returned. In order to put a check on the secret destruction of life by poison in this country, analyses of the contents of the stomach should be more frequently made. Impunity in one instance, as a result of a careless inquiry, uniformly leads to the perpetration of a series of murders.

Rules for conducting an analysis.—Before proceeding to the analysis of any suspected substance, we should, if possible, make ourselves fully acquainted either with the symptoms or appearances, or both, observed in the person suspected to have been poisoned. We may by a knowledge of these facts determine, à priori, whether we shall have to search for a neurotic, irritant, or corrosive substance. The kind of poison may often be predicted from the symptoms and appearances, and our analysis directed accordingly. I have, however, known more than one instance, where an irritant poison has been sought for in the contents of the stomach, when every fact connected with the death of the party, as well as the rapidity with which death took place, tended clearly to show that if any poison had been used, it must have been one of the pure narcotics. It is not unusual to find the examination of medical witnesses misconducted in courts of law, in relation to the effects of, as well as the tests for poisons. The deceased may have died from a narcotic, while questions relative to the action of irritants alone, will be put by the counsel for the prosecution and defence, or *vice versa.*

The chemical evidence may be divided into several branches. The analysis may extend—

1. To the pure poison. We may be required to state the nature of a substance (part of the poison administered) found in the possession of a prisoner, or lying near the deceased.

2. The analysis may be confined to a portion of the substance of which the affected party partook; and here the poison is usually mixed up with liquids or solids of an organic nature. The steps of the analysis are then rather more difficult. *a.* There may have been various substances combined in a meal, and the poison have been mixed with one substance only. This will show the necessity for examining separately the various articles used at a meal, if we wish to discover the real vehicle of the poison. *b.* Symptoms of poisoning may occur after the eating of a pudding. A part of the pudding may be analyzed, and no poison discovered; because the poison, instead of being incorporated with the dough, may have been loosely sprinkled like flour over the exterior only. *c.* A similar circumstance may occur in the poisoning of a dish of meat. The gravy may be poisoned, and not the meat. A case of this kind occurred to Dr. Christison. A whole family was attacked with symptoms of poisoning after a meal on roast beef. The meat was examined, but no poison could be discovered. It was then ascertained that the poison had been mixed with the gravy, and those who had taken the meat without the gravy suffered but slightly. In one instance, referred to me, arsenic was placed instead of salt on the edge of the plate of the deceased. (The Queen v. Jennings, Berks Lent Assizes, 1845.) No other person experienced symptoms of poisoning after the meal, except the child who ate out of that plate. In the case of Bodle, tried in 1833, the deceased was proved to have been poisoned by arsenic administered in coffee. The coffee was kept ground in a bottle, to which every one of the family had access; and there could be but little doubt, from the circumstantial evidence, that the poison had been mixed with the coffee in this bottle. That which remained in the bottle was carefully examined by the late Mr. Marsh, but no trace of arsenic could be detected. The poison had most probably been mixed with the *upper stratum* only of the powdered coffee, and the whole of the poisoned portion had been used for breakfast. A remarkable fact was brought out in the case of the Queen v. Edwards (Central Criminal Court, November, 1844). The deceased, it was stated, had died from drinking part of the contents of a bottle suspected to contain sugar of lead,—but it was proved that some of the same liquid had been drunk by another person the night previously without any injury resulting. The medical witness explained this by saying that the poison existed as a crust in the bottle, which might have been detached in one case and adherent in the other. A somewhat similar case is given under the section on CARBONATE OF LEAD. (See that compound, post.) Facts of this kind are of some medico-legal importance: they will often enable a witness to explain certain anomalies in cases of poisoning. By bearing them in mind, it is easy to understand, how it is that one or two persons only

will suffer at a meal made in common or on the same article of food, while others will escape.

3. The chemical analysis may be directed to the matters *vomited* and evacuated. In irritant poisoning, a large quantity of poison is often expelled in this manner, and may be detected especially in the matter first vomited. In a suspected case, an immediate analysis should be made of the matters ejected from the stomach. They may be regarded as furnishing to the medical jurist the proofs required to establish the *corpus delicti.*

4. If death has ensued, an analysis of the contents of the stomach and intestines must be made. Supposing no vomiting to have occurred, or that this has been slight, and death has taken place speedily, then we may expect to find abundant traces of the poison in the viscera. If no poison should be found in the stomach,—the contents of the duodenum and the other small intestines, as well as of the rectum, must be separately examined. If the poison cannot be detected in the contents of the stomach and intestines, it must be sought for in the tissues of the viscera, especially of the liver and spleen.

As a summary of the various conditions under which poison may be discovered by chemical analysis, it may be stated, that in the *living* body, chemical evidence is derivable from an examination: 1, of the matters vomited; 2, of the evacuations; and 3, of the urine: in the *dead* body poison may be found *free*, 1, in the stomach; 2, in the small intestines; 3, in the rectum;—or *absorbed;* 4, in the blood; 5, in the liver; 6, in the spleen and kidneys; 7, in the heart; 8, in the lungs; 9, in the muscles.

It is obvious that one or several of these sources of chemical evidence may be wanting, and it is rare in any one case of criminal poisoning that all are open to a medical witness. The detection of poison in the vomited matters during life, and in the stomach, intestines, liver, or other organs after death, is of course the most satisfactory kind of chemical evidence; since, *cæteris paribus*, it is a clear proof of poison having really been taken. It is difficult to admit the supposition that it should have been designedly introduced after death; besides, in such a case, the absence of all marks of vital reaction, and of any symptoms during life indicative of poisoning, would remove such a suspicion. If the poison be detected in the tissues of any of the organs, and due allowance be made for imbibition from adjacent organs (see ante, page 68), there can be no doubt of its having been introduced into the body during life. The presence of poison in the stomach and bowels, or their contents, with such marks of vital reaction as are known to be produced by the particular substance, as for instance, inflammation in the case of the irritants, affords the strongest presumptive evidence of death from poison, open to be rebutted by other proofs of death from disease, under which the deceased may have been laboring at the time.

Causes of the non-detection of poison.—But let us take the case, that chemical evidence is entirely wanting, and that no poison is detected under any of the circumstances mentioned; if there be other facts to render death from poisoning probable, we must endeavor to explain why this important branch of evidence has failed. There are few medical jurists who have not met with cases in which, although undoubtedly death was occasioned by poison, either irritant or narcotic,—not a trace of the noxious substance could be detected in the solids or liquids of the body. The non-discovery of poisons in cases of poisoning may depend:

1. *On the nature of the poison.*—In the present state of our knowledge, chemistry, with few exceptions, furnishes us with the means of identifying with certainty in the dead body a mineral poison only. The greater number of vegetable poisons, when diffused through the body, are beyond the reach of chemical analysis. Botanical characters may sometimes serve to point out the nature of the substance; but only in those instances in which the plant has been swallowed in the state of leaves, roots, or seeds. If the extract, or inspissated juice, has been administered, or if the poison were in the form of infusion, tincture, or decoction, a chemical analysis will commonly be of no avail. The same remarks apply to the powerful alkaloids extracted from vegetables. It is true that there are delicate color-tests for morphia, strychnia, veratria, and a few others; but unless the poison can be obtained in an isolated state, so that its principal character may conclusively be determined, these are not satisfactory as a basis for chemical evidence in cases of criminal or casual poisoning. Some poisons admit of no known means of detection by chemical analysis. A few slices of the root of the œnanthe crocata will destroy life in two hours. No poison of any kind has hitherto been separated from this plant. A similar remark may be made of the Ordeal bean of Africa, and of the decoction and infusion of the bark of laburnum. Although doubtless some poisonous ingredient of a very active kind is absorbed and diffused with the blood in these cases, no chemist has ever pretended to extract the subtle agent. Like the poison of rabies and of the viper, it is at present beyond the reach of chemical science. It is to be regretted that the facts connected with the chemical evidence of poisoning, have been placed by some "experts" in a false light before the public. Professor Casper, of Berlin, in his recently published work (Handbuch der Ger. Med. 1857, p. 395), correctly observes, that modern chemistry, with all its progress, has left and must leave many problems unsolved in reference to the chemical analysis of the parts of a body dead from poison. "There are many poisons,—for example, the alkaloids,—which chemical processes cannot discover in the body. Fortunately for medico-legal practice in Germany, these forms of poisoning do not often present themselves to the chemist or medical jurist. But there is another reason for exposing that popular error which over-estimates the value of chemical evidence in these cases. Every one who has studied the writings of chemists, must have perceived how widely the highest authorities differ from each other as to the best processes for the detection of poison. Those who are practically informed in these matters, know, that the method of analysis pursued by one, is condemned by another as inefficient. These considerations have induced me, and must induce every medical jurist, to include as a necessary part of the evidence of poisoning, a combination of all the circumstances connected with the illness and death of the deceased." In this country, we have recently had the strange spectacle of men swearing to the efficiency of their own processes of analysis, each being different from the other,

and of their being openly allowed to condemn those pursued by witnesses who differed from them in opinion. If a criminal court is to take upon itself a judgment on such scientific matters, it should at least allow a right of reply to those whose processes of research are attacked. The public generally adopt that view, whether right or wrong, which may tend to save the life of a criminal.

Poisons, that are of a highly *volatile* nature, may be speedily dissipated; so that in a few hours, or a few days after death, none may be discovered. Alcohol is well known to pass away so rapidly, that no spirituous odor may be perceived in the contents of the stomach, although the person may have died speedily, and the body be inspected within six or eight hours after death. See Exp. by Dr. Percy (ante, p. 80), in which three ounces of alcohol had entirely disappeared in eight hours. Prussic acid, chloroform, and nicotine, may be in like manner rapidly lost or decomposed. (See PRUSSIC ACID.)

2. *Influence of vomiting and purging.*—The non-detection of poison in the viscera, may be owing to its having been expelled by excessive *vomiting and purging.* In all such cases, however, the poisonous substance ought to be found in the vomited matters, if these are forthcoming. In two instances of poisoning by sulphuric acid, in two of arsenic, and in one of oxalic acid, although death took place with the usual rapidity, not one of these poisons existed in the stomach of the deceased. Similar cases are to be found reported in most works on medical jurisprudence. (See cases of Humphreys, ante, p. 113, and Alexander, p. 49.) It may, however, be fairly inferred that, in all cases of irritant poisoning, where the vomiting and purging have been slight, some portion of the poison ought to be found in the body, if the individual have died within the average period, *i. e.* if he have not survived more than two or three days. Should none be present under these circumstances, it may be a question, whether death was really due to poison. It is not probable that a common dose of arsenic would be entirely removed by absorption in the course of two or three days. (See the case of the Queen v. Hunter, Liverpool Lent Assizes, 1843, ante, p. 49.) Vomiting and purging cannot affect that portion of poison which has been absorbed and deposited in the organs. The quantity of poison actually taken by deceased, has not so much influence on the power of detection as the quantity which remains in the body at the time of death. This is subject to great variation.

3. *Loss by absorption and elimination.*—Solid mineral poisons are usually detected without difficulty, because they are generally administered criminally in large doses; but in cases of chronic poisoning, *i. e.* where the substance is administered in small doses at long intervals, chemical analysis will sometimes fail: for the poison may become entirely absorbed and eliminated. Orfila has discovered that arsenic and antimony are especially liable to be excreted with the urine; arsenic is also eliminated in the bile and in the serous exhalation of the chest. Thus, if the dose of arsenic be small, if the poison be taken in a state favorable for absorption, *i. e.* in solution, or the person survive for a period of two or three weeks, no trace of the substance may be found in the body. (See ante, Elimination of Poisons, p. 49.)

It cannot be denied, that the great facility with which chemical analysis is applied to the detection of most mineral poisons, is due to the ignorance of those who criminally administer them. A mineral poison is frequently given in the form of a loose powder, undissolved; and it is then easily susceptible of analysis. Instances of extraordinary depravity have, however, occurred, in which persons have shown themselves to be acquainted with these facts, and they have endeavored so to destroy their victims, as to frustrate the usual means of detection. A case was tried at Mayence, in which the evidence clearly proved, that the prisoners had poisoned the deceased and several persons previously, by administering to them arsenic in a saturated solution in water. One of them confessed that she had boiled the poison in water, allowed it to cool, filtered the solution, and then administered it by small quantities at a time in wine, milk, gruel, and other liquids. On one of these occasions the dose of poison happened to be so large, that it operated fatally,—a circumstance which led to the detection of the crime. As it might have been anticipated, not a trace of arsenic could be discovered on analyzing the contents of the viscera of those who had perished in this manner. A case somewhat similar is related by Dr. Christison to have occurred in Scotland. (Op. cit. p. 319.) The celebrated Aqua Toffana appears to have been a pure solution of arsenic.

I have elsewhere (ante, p. 49), adverted to the case of Hunter. The time required for the expulsion of arsenic from the body was here material. The deceased survived three days, and had suffered during the whole time from vomiting and purging. No arsenic was found. The state in which the poison was supposed to have been given, was not in evidence. It was alleged to have been administered in milk, which the counsel thought might have acted as an antidote to it; but this would not have affected it chemically, or have prevented its absorption. It is possible that violent vomiting and purging at intervals, for less than three days, may entirely carry off this poison from the contents of the stomach and bowels. I have met with several instances in support of this rapid expulsion of large doses of arsenic (G. H. Rep. April, 1837, p. 79); but had an analysis been here made, *absorbed* arsenic might have been discovered in the tissues, supposing it to have been administered and to have caused death. The exact time which is required for absorbed arsenic to be entirely removed from the body by elimination is undetermined (ante, p. 49). I have elsewhere stated, that the French medical jurists assign a period of about fifteen days (survivorship) for its entire disappearance from the human body, when taken in an ordinary dose (see p. 51). That this is not far from the truth is proved by the case of Dr. Alexander (see ante, p. 49). This gentleman died in sixteen days from the effects of a large dose of arsenic. Not a trace of either free or absorbed arsenic existed in the body. Alcohol and prussic acid may also entirely disappear by absorption, and by elimination through the lungs. An instance was communicated to me in which a man by mistake swallowed forty-five drops of prussic acid of a strength of two per cent. He lay insensible for four hours: he then vomited and recovered. The vomited matter had no odor of the poison. In the case of Ballet (see Morphia, post), although a large dose of acetate of morphia had been given to the deceased, no trace of this poison was discovered. The poison is of a soluble kind: there had been much vomiting and purging, and deceased survived thirty hours. (Guy's Hosp. Reports, Oct. 1856, p. 321.)

4. *Decomposition of the poison in the living body.*—It has been frequently a subject of remark that in poisoning by opium, if the person survive many hours, no trace of the substance, either as opium, morphia, or meconic acid, has been found in the stomach, bowels, or tissues. Several cases of this kind have occurred to Dr. Christison (On Poisons, 4th edit. p. 697), and others of a similar nature have occurred to myself. In a case of death from arsenic in April, 1858, I had an opportunity of again examining this question. The deceased took, twelve hours before death, five grains of calomel with one grain and a half of opium; and four hours before death, two grains of calomel and one and a half grains of opium, making three grains of opium and seven grains of calomel. Mercury (from calomel) was found in large quantity in the stomach and duodenum, but there was no trace of meconic acid or morphia (opium). There was no mercury in the lower bowels.

In August, 1857, I was consulted respecting a case of alleged poisoning by opium, which was the subject of a trial for murder at the Liverpool Assizes. A child died under the usual symptoms of narcotic poisoning, and it was proved that the mother, who was charged with the murder, had procured from a druggist, on the day before the death, one hundred drops of laudanum. On inspection, there was congestion of the brain and its membranes, but no natural cause of death. Mr. Stone, of Manchester, made an analysis of the stomach and viscera. No morphia—the poisonous alkaloid of opium, was found anywhere; but from one result Mr. Stone was led to suspect the presence of meconic acid. He declined, however, to speak positively to this, or to state that the result unequivocally proved the presence of opium in the stomach. Although there could be no doubt, from the medical evidence, that death was caused by opium, no morphia could be detected in the body.

Prof. Buchner has observed that chemical analysis has failed to detect opium where it had been certainly administered in large quantity. Apart from the influence of treatment by emetics, the stomach-pump and purgatives, which may mechanically remove many poisons from the body, there must be other causes to account for the frequent disappearance of this poison. It has been supposed to undergo a change in the stomach like other organic substances; and the morphia which is absorbed, is believed by some to be so changed in its nature that it cannot be detected in the tissues like mineral poisons. (See Guy's Hosp. Reports, Oct. 1856, p. 129.) This theory of partial decomposition has been also propounded with respect to strychnia. At the trial of Palmer it was contended by some of the witnesses that no person could die from strychnia without a portion of the poison remaining in the body; while others affirmed that there might be circumstances which would prevent this detection in every case,—and that removal by absorption and partial decomposition were among these circumstances. It is quite certain, from facts since published by Mr. Horsley, Dr. De Vry, Dr. Crawcour, and others (see ante, p. 76), as well as from observations since made by

Drs. Christison, Maclagan, and Geoghegan, that in some cases of undoubted death from strychnia, the poison is either not present in the tissues at all, or its condition is so changed that it cannot be recognized by its usual properties. These facts are in strict accordance with those which experience has already settled for other poisons.

The following case shows that phosphorus may entirely disappear from the body: A trial took place at the Bodmin Assizes (Reg. v. Beard, July, 1857), in which the accused was charged with the wilful murder of his grandchild, by poisoning it with phosphorus. It was proved that the prisoner had procured a pot of phosphorus-paste, as it is used for destroying vermin, on false pretences; and the evidence went to show that he had administered this paste on bread to the deceased, at various times. The child died on the fourth day (April 5th), under such symptoms as phosphorus would produce. The body was buried on the 6th, and exhumed for inspection and analysis on the 29th. Mr. Herapath, of Bristol, stated in his evidence at the trial, that he found the stomach highly inflamed and blistered, the membrane raised in small vesications. The duodenum was not inflamed but blistered. The remainder of the intestines were generally in a natural state, except that there was intussusception at one part. The lower part of the intestines was blistered. He referred death to irritant poison, and he considered that poison to be phosphorus. *He could not find any trace of it in the body.* In order to account for its absence, he propounded this theory: "Assuming the child to have been poisoned by it, I say that phosphorus is constantly being destroyed by the action of the air, slowly burning and passing into phosphorous and phosphoric acids; and I should expect, after the sickness, that the small quantity (?) of phosphorus which remained during a month's exposure in the grave, and in the opening of the stomach, would be changed into those acids." As these acids are not volatile, they would, if thus formed, have remained in the liquids, not to mention that phosphorus is known to chemists by a peculiar odor, even when existing in very small quantities. Hence it is to be inferred that the best available means were used by Mr. Herapath, and no phosphorus, and no products of the decomposition of phosphorus, were found! There can be no doubt that if phosphorus was there, Mr. Herapath ought to have found it; but not being there, it would not be justifiable to infer that the child had not died from the effects of phosphorus, although such an inference might receive support from the chemical evidence which this gentleman and others gave for the defence at the trial of Palmer. In fact, Mr. Herapath found it either necessary or expedient in this case, to lay aside chemistry as altogether inadequate to solve the question, and to base his opinion on the symptoms and appearances in the body!

It is quite certain that some poisons do undergo a change in the blood, by which their nature is so altered that they can be no longer recognized by the usual tests. Drs. Christison and Coindet could not discover any oxalic acid in the blood of the veins (vena cava) of a dog which had died in thirty seconds from the actual injection of eight and a half grains of that poison into the femoral vein (On Poisons, 4th edit. p. 18), and I believe that he has furnished a true explanation of this fact in stating that certain poisons "either cause obvious changes in the constitution of the blood, and themselves undergo alteration likewise; or without the blood becoming appreciably different in its properties from the healthy state, the poison undergoes a rapid change in the molecular affinities of its elements, and so disappears" (ibidem). If the quantity of poison absorbed be small, it may wholly disappear; if large, a portion may still be detected. This observation has been made with respect to the oil of bitter almonds (see ante, page 87).

Most organic poisons are used as medicines; and their beneficial operation as such is supposed to be owing to changes which they produce in the blood and tissues. It is hardly probable that they should produce such changes without themselves undergoing some chemical change. The non-detection of certain organic poisons, such as strychnia and morphia, in the tissues and secretions of human beings and animals poisoned by these alkaloids, renders it probable that they undergo some change within the system. The allegation that they have been found in the tissues and urine in certain cases, does not at all affect the negative results above mentioned.

5. *Influence of the quantity taken.*—The power of detecting poison in the dead must depend, not on the dose taken, but on the quantity remaining in the stomach and other organs at the time of death. However large the dose, if the person has survived some hours or days, the residual quantity may be very small. Again, the dose originally taken may have been so small, that by the mere effect of diffusion, it may be difficult, if not impossible, to trace it. I have elsewhere published the case of a child that died in four hours, from the sixteenth part of a grain of strychnia, and that of an adult who died in twenty minutes, from half a grain (Guy's Hospital Reports, Oct. 1856, p. 138). It must be obvious that these small quantities, absorbed and diffused through the whole of the body, would be beyond the reach of such a chemical analysis as would or ought to satisfy a court of law on a charge of murder. Assuming, in the case of the adult, that the half grain was entirely absorbed, and that it remained unchanged in the blood, the proportion of strychnia in a pound of that blood would not exceed the fiftieth part of a grain, or one eight-hundredth of a grain in an ounce! In the case of the child, if none were eliminated or unchanged in the body, the proportion would be only the one two-hundred and fortieth of a grain in a pound of blood, or about one four-thousandth of a grain in an ounce! It is true that the power of infallibly detecting by chemical processes, quantities smaller than these, has been claimed by certain "experts," but the claimants have carefully concealed from the public and profession, that their experiments referred to the pure poison *out of the body*, and not to such small quantities diffused through the dead blood and tissues. The one-thousandth of a grain may be, in a certain sense, detected out of the living body; but no expert has yet given this quantity to an animal, and separated it again, or any part of it, from the blood. By one chemist, strychnia is said to be eliminated in the urine; by another, to be deposited in the flesh and bones; by a third, to remain stationary so as to be entirely recoverable after having done its work as a poison; and by a fourth, to be speedily thrown out

of the system. These inconsistent statements show that there is a total want of uniformity in the results obtained by different analysts.

In the accidental poisoning of infants by opium, the fatal dose is frequently so small that there can be no reasonable expectation of finding any morphia or meconic acid, either in the absorbed or unabsorbed state. In one instance in which I was consulted, a child, six years of age, died in thirty-six hours, from three-quarters of a grain of opium, given in divided doses. The quantity of morphia which killed this child, would not, at a maximum, have exceeded the one-tenth of a grain. Not a trace either of this alkaloid or of meconic acid was found in the body.

Infants have been frequently killed in a few hours by doses of opiate preparations corresponding to the fifth, the eighth, the tenth, and even the twelfth part of a grain of opium, equivalent, in the first case, to one-fortieth of a grain, and in the last case, to one ninety-sixth of a grain of morphia! Dr. E. Smith has lately recorded a case in which an infant, seven days old, died comatose eighteen hours after having taken *one minim* of tincture of opium. Symptoms of narcotic poisoning set in, in about half an hour, and the child never rallied from the effects of the drug. Here not more than one-twelfth of a grain of opium, corresponding to the hundred and twentieth grain of morphia, destroyed life (Medical Times and Gazette, April 15th, 1854, p. 386). Is it to be supposed, in reference to such cases, that even if there were no absorption and diffusion of the morphia through the body of the child, a chemist could separate this minute quantity of morphia from the stomach! If a man ventured to assert this, no one who knows practically the properties of opium would believe the assertion. The doctrine that morphia must in all cases be found, before death is attributed to opium, would lead to wholesale infanticide, without any chance of proving the perpetration of the crime.

When the poison is of a nature to be easily detected, the quantity taken may have been so small that none will be found after death. Dr. Edwards has reported the cases of five children poisoned by colored sweetmeats; the poison had been taken in very small quantity and in repeated doses. Three died, but arsenic was found only in the stomachs of one of these. (Pharm. Journal, February, 1857, p. 417.)

6. *Decomposition and loss of the poison in the dead body.*—Although poisons may remain in the body at the time of death, still some are liable, either to become altered, or to disappear during the progress of the putrefactive process. Soluble poisons contained in the viscera are soon lost. I have found in the stomach no trace of a large dose of oxalic acid given in solution to an animal which had been buried twenty-eight days. Some organic poisons easily undergo decomposition; while mineral poisons resist change. Thus it is well known that arsenic, if taken as a solid, may be found after many years. How long absorbed arsenic may remain in the soft organs it is not easy to say. I have found it after two years' interment; but the result of an analysis must depend materially on the quantity which happens to be retained by the organs at the time of death. If this should have been small, it may not be discovered when the body has been buried for a long period.

In general, the residuary arsenic found in the stomach or bowels of a putrefied body, is partly converted to yellow sulphuret, or orpiment. This substance dyes deeply the coats of the stomach or intestines, so that yellow spots may be sometimes seen on the outside. The diaphragm and other viscera in contact with the stomach are also stained yellow. The sulphur resulting from the putrefaction of the soft parts produces this conversion; but it is never complete. The indigo or soot with which arsenic is now colored, is not liable to change; and it is proper, when arsenic is found in a stomach, that the coloring ingredients should be looked for. The proportion of coloring matter mixed with arsenic is always small, amounting to about three per cent. for indigo, and about six per cent. for soot. Being lighter than the mineral it may be washed away, and carried off by vomiting and purging during life; hence, although arsenic may be found, the coloring matter may not be present. In one case in which indigo-arsenic, and another in which sooty arsenic had been taken, no trace of the coloring matter was found in either stomach, although arsenic was present in the proportion of several grains.

The salts of iron are generally found in a putrefied body in a state of black sulphuret. Late researches have shown that strychnia is not destroyed or materially changed in its properties by putrefaction (Guy's Hosp. Reports, Oct. 1856, p. 393), and this property of resisting decomposition may apply to other pure alkaloids. With respect to prussic acid, its great volatility must be a bar to its detection in the body, for a long period after death. The vapor of this acid easily traverses wet or dry animal membranes, and in contact with ammonia and sulphuretted hydrogen, as results of putrefaction, it may be transformed into sulphocyanide of ammonium, and its usual properties destroyed. According to Mr. Herapath it is by reason of this conversion that prussic acid rapidly disappears in the dead body, and he advises that sulphocyanic acid, or a sulphocyanide, should be sought for in the blood. In a case of poisoning by oil of bitter almonds he states that *two months* after death he found no trace of prussic acid in the stomach or intestines, but upon analyzing the blood, although he could not prove the presence either of oil of almonds or free prussic acid, he was able to detect a small quantity of hydrosulphocyanic acid, or an alkaline sulphocyanide; and from this he came to the conclusion that the deceased had poisoned herself with prussic acid, oil of bitter almonds, laurel water, or some other similar compound! (Chemist. 1854, i, 321.) As, however, in this case there was no prussic acid in the stomach, intestines, or blood, it was a pure speculation to refer the presence of traces of sulphocyanic acid in the blood to the decomposition of prussic acid taken two months before! If the poison had entirely escaped from the stomach, it is far more probable that it had escaped from the blood. A reliance upon the discovery of a sulphocyanide under such circumstances, might give rise to a dangerous fallacy; this compound naturally exists in the saliva; it may find its way into the stomach, or be a product of decomposition in the blood. Nothing but the discovery of prussic acid itself should be considered as furnishing satisfactory evidence.

OBJECTS OF A CHEMICAL ANALYSIS.—A chemical analysis is commonly directed in toxicology to the determination of the following points:—1. The *nature* of the poison. 2. The proportion, or *quantity*, in which it has been taken. 3. The solution of certain questions connected with the criminal administration of poison.

1. The *nature* of the poison and the probable quantity administered, are usually stated in an indictment; but it is not absolutely necessary for conviction that the substance thus stated should be proved to have been that which was actually administered. There were some medical difficulties formerly connected with this subject, since, on an indictment for poisoning, it was always necessary to prove that death was caused by poison; but the person may be indicted for murder, and the proof of the means of death is not now indispensable. By the statute 14 and 15 Vict. c. 100, s. 4, it shall not be necessary "to set forth the manner in which, or the means by which, the death of the deceased was caused; but it shall be sufficient in every indictment for murder, to charge, that the defendant did feloniously, wilfully, and of his malice aforethought, kill and murder the deceased." It must be shown that the substance or substances administered were of a noxious or poisonous nature, and either caused or accelerated death.

2. The *quantity* of poison administered is generally stated conjecturally; but when any portion of the original vehicle of the poison is discovered, it is in the power of a witness to give a tolerably accurate opinion of the quantity taken. Thus, all solid substances given for analysis should be first weighed, and all liquids measured: a quantitative analysis may then be performed at any subsequent period. The chief question in law in regard to the quantity of poison is, whether it was sufficient to destroy life, or to produce any serious effects? The malicious intention of a prisoner is often to be inferred from the quantity of poison existing in the substance administered. A case occurred some years since, in which a man was capitally indicted for administering oxalic acid with intent to murder. The poison was introduced into coffee, served for the prosecutor's breakfast. There could be no doubt of its presence; but on estimating the quantity, Mr. Barry discovered that it was only in the proportion of about ten grains to a pint, a quantity which he considered insufficient to produce any serious effects on the body. The prisoner was acquitted; but it is obvious, that had the proportion been an ounce to a pint, the malice of the act would have been apparent. This case shows that a medical jurist must not be content with merely determining the presence of poison in suspected liquids,—he should also determine the quantity. The law presumes upon the innocence rather than upon the guilt of an accused party, when the evidence fails in showing, from the small quantity of the substance administered, that the act was malicious. If a man gave to another a few drops of sulphuric acid in a large quantity of water, we should not infer that his intention was to murder; but if he administered a large quantity of the acid in an undiluted state, the malice of the act would be at once apparent. Presumptions of this kind must, of course, be affected, as well by the nature of the poison, as by the moral circumstances adduced in evidence. A prisoner has sometimes alleged in his defence, that he did not know the substance to be a poison, and that he did not administer it with intent to kill. The law, however, properly infers

that the highly destructive properties of such substances as arsenic or corrosive sublimate, must have been well known to the prisoner, if an adult, by common repute.

It need hardly be observed, that the *quantity remaining in the stomach*, or the portion of *absorbed* poison deposited in the tissues, can give no idea of the quantity actually taken by the deceased: since more or less of the poison may have been removed by violent vomiting and purging as well as by elimination. But the quantity found free in the stomach and bowels, even after a portion has been thus lost, is often more than sufficient to destroy the life of a human being. It is singular that, notwithstanding the existence of these very obvious and natural causes for the removal of a poison from the stomach, barristers should so frequently address to a medical witness—whether the quantity of poison found in the body was sufficient to cause death? Whether this question be answered in the affirmative or negative, is a matter which, medically speaking, cannot at all affect the case, since either no traces of poison, or but a very small portion, may be found in the viscera, and yet the deceased may have assuredly died from its effects. Absorbed arsenic, as it exists in the tissues, is never found except in very minute proportion, a proportion commonly insufficient to destroy the life of another. (See ante, p. 46.) Hence, whether much or little be detected, the object of this question is not very apparent; since the fact of death having been caused by poison does not, in the least degree, depend upon the precise quantity which happens to remain in a dead body. It has been truly remarked by Orfila, in regard to arsenic, and it equally applies to all poisons, that that portion which is found in the stomach *is not that which has caused death;* but the *surplus* of the quantity which has already produced fatal effects by its absorption into the system. The inquiry should, therefore, be directed to the probable quantity of poison *taken;* not to how much remains in the body.

This question is one of more importance than may at first sight appear. There is scarcely a trial for criminal poisoning, in which it is not put to a medical witness, either by the judge, or the counsel for the prosecution or defence. Supposing poison to be found in the stomach, but not in sufficient quantity to destroy life,—is it therefore to be assumed that the person did not die from its effects? This would be equal to laying down the doctrine, in the face of the most indisputable evidence to the contrary, —that poisons, when taken into the body, are never liable to be expelled by vomiting or purging, or to be removed from the stomach by absorption and carried out of the body by elimination. The real object of a toxicologist is to discover the poison by clear and undoubted evidence. If more than sufficient to cause death be found in a dead body, then the dose must have been larger than was necessary; but if this proof be always required, what is to become of those cases of criminal poisoning in which the prisoner administers a dose only just sufficient to destroy life, or in which the deceased, by the strength of his constitution, happens to survive the effects for some days or weeks, and ultimately dies of exhaustion? No poison would be detected under these circumstances. (See the case of Dr. Alexander, ante, p. 49.) Orfila has most completely demonstrated the fallacy of this objection to medical evidence, and the danger of a court of law relying upon it. (See Ann. d'Hyg. 1845, i, 347; also Toxicologie, ii, 781.)

As an illustration of the kind of cross-examination which a medical witness must be prepared to undergo on this question, I subjoin an extract from the Report of the trial of Reg. v. Palmer (May, 1856). Sergeant Shee, in directing this question to me, thus deals with the matter:

"Q. Have you not told me, that the quantity of antimony that you found in Cook's body, was not sufficient to account for death? A. Perfectly so; but what was found in Cook's body was not all that he took; if a man takes antimony—Q. Do you wish to add to your testimony? A. I do, because I see, it is only a little misunderstanding; if a man takes antimony, it produces these effects: first, he vomits, by which some passes out of the body; some may escape by the bowels; there is a great deal that passes off at once by absorption, and is carried out with the urine. I find, by the experiments of Orfila, upon whom we are all inclined to rely, that in from four to seventeen hours, antimony is found passing out by the urine.—Q. Do you mean, on your oath, to say, from such traces of antimony as you found in Cook's body, you were justified in stating that your opinion was that his death may have been caused by antimony? A. Positively and decidedly so; the amount found in his dead body affords not the slightest criterion of what he may have swallowed while living. I have sometimes found in a body less arsenic than would account for death.—Q. But, if the amount found is not the slightest criterion of what may have been administered, how does that justify you as an analytical chemist, in stating your opinion that so small a quantity may have caused death? A. I have not said what quantity may have caused death. I have said a certain quantity was found in his body, which may have been the residue of what had caused death." (The Queen v. Palmer, Report of Trial, p. 143.)

The fallacies connected with this line of examination must be apparent. In no death, from antimony, yet recorded, has such a quantity of this substance been found in a body as would suffice to kill another person! When given in divided doses,—as the evidence proved that it had been given in the case of Cook,—and there has been violent vomiting in the intervals, it is not probable that small doses would accumulate and remain in the stomach and intestines for a week. Such questions, therefore, were only calculated to conceal the truth, and mislead the jury.

The fallacy based on this inquiry, is not confined to lawyers. Some medical men, even of good professional standing, have paid so little attention to the subject of toxicology that, if the quantity of poison remaining in a dead body were less than that which is usually described as a *fatal dose*, they would be prepared to say that death was not caused by poison. They expect either that the whole dose swallowed should remain in the body as evidence against the administrator, or that vomiting, purging, and absorption, are so nicely adjusted that, to meet their theory, these functions are wholly arrested when the quantity is reduced to a minimum fatal dose. The half-grain of antimony found in the body of Cook, might, however, be taken to represent the residue (at the time of death) of ten, twenty, or one hundred grains of tartar emetic taken during life! In spite of this obvious inference, Sergeant Shee was allowed, on this occasion, to mystify the facts and to place the matter before the jury, as if the half-grain found in the dead body was the whole quantity of antimony that deceased could have taken, and, as this residue did not amount to a fatal dose for another person, it was insufficient to account for the deceased's death! Some of his medical advisers appear to have adopted the same view, since they represented it as a question whether half a grain of antimony could or could not account for the death of the deceased. Either ignorantly or designedly, they entirely overlooked the fact, that there had been severe vomiting at intervals some days before death, and that such vomiting could not take place without the expulsion and loss of a portion of the substance taken.

When the *quantity* of poison found in a dead body, either in the free or absorbed state, is *small*, it is, of course, a fair question, whether it may not be the remains of doses given medicinally, and without criminal intention. Arsenic and antimony are frequently used in medicine; and, if a person dies while taking Fowler's mineral solution, or solution of tartar emetic, one or both of these metals may be found in the stomach, or deposited in the liver. I am informed that, in the Fen districts, Fowler's solution of arsenic is much used by the poor as a preventive of ague; and that they readily procure it from druggists for domestic use, without a medical prescription. Accidents sometimes occur to children by reason of an overdose, given with innocent intention. Small quantities of mercury are not unfrequently found in examining the livers of persons who have died after medical treatment. The taking of a dose of calomel, or blue pill, shortly before death, may account for this. In the incineration of a liver, a small quantity of copper, or lead, may be discovered,—introduced accidentally during life. Opium, morphia, strychnia, or prussic acid, may be thus found in the body of a person who has died suddenly while taking any one of these poisons medicinally. We are bound, therefore, to consider before we place any reliance on such chemical evidence as "minute," "distinct," or "unequivocal traces" of poison, whether medicinal use or accidental introduction may not account for the discovery. Who prescribed the medicine? For what was it prescribed? What were the symptoms preceding death? When the case is really of that innocent complexion which some barristers are inclined to assign to it, from the small quantity of poison found, there will be no difficulty in obtaining answers to these questions. In Wooler's case (ante, p. 108), the medical men did not prescribe arsenic, and the deceased, at least some time before she died, could not have access to arsenic. In Cook's case the medical men, called in by the criminal, did not prescribe antimony, and would not have prescribed it medicinally, under the circumstances. Deceased had no access to antimony, and there was no reason for his taking it secretly. He was sick when the murderer was about him, and the sickness abated in his absence. The presence of even a small quantity of antimony in his dead body, therefore, at once explained *that* for which no other theory could satisfactorily account.

We may now take the converse proposition,—a *large quantity* of poison may be found in a dead body, both in the *free* and *absorbed* state. What then is the inference? Under these circumstances, no theory of medical treatment, or of the innocent use of medicine, would be applicable. In a case of this kind, the cause of death may admit of no dispute; not because the residuary quantity of poison is large, but because the symptoms under which the persons suffered, and the appearances, may be such that there is no room for doubt. A medical man must remember that a large quantity of poison may be injected into the stomach or rectum after death. Such a state of things would be indicated by the absence of symptoms and appearances. These contingencies tend to show how important it is that we rely not too strongly or implicitly on mere chemical results. The discovery of absorbed poison removes any difficulty in respect to injection after death, and proves that the poison entered the body during life, provided we have satisfied ourselves on the conditions for cadaveric imbibition, elsewhere pointed out (ante, page 68). The quantity of residuary poison found in a dead body has been supposed to throw a light upon another important branch of medico-legal inquiry; namely, whether the act of poisoning was one of *murder or suicide.* A large quantity is considered to indicate a conscious and deliberate swallowing of the poison, and under certain limitations this is no doubt correct. When the poison is either in a liquid or solid form, and when it has a very strong taste and odor, it is difficult to come to the conclusion that it could have been taken unknowingly. In the case of Mr. Sadleir there was reason to believe, from the large quantity of essential oil of bitter almonds found in the body, that the deceased must have swallowed many ounces. A large dose of this oil could not be taken unknowingly. In the section on Arsenic will be found a case in which a man, æt. twenty-two years, swallowed from four to six ounces of arsenic. He died in four hours, having suffered from vomiting and purging. After death, *two ounces* of arsenic were found in his stomach. The poison may be poured down the throat while the person is sleeping, as in the case of Humphreys (ante, page 113), or it may be forcibly introduced into the mouth, as in the case of Fougnies, for whose murder the Count Bocarmé was executed in Belgium; but these modes of administration invariably lead to detection.

The question arising from the discovery of a large quantity of arsenic in the stomach, was first seriously raised in the case of Madeline Smith (Edinburgh Court of Justiciary, July, 1857). The deceased, L'Angelier, died from the effects of arsenic under the usual symptoms of vomiting and purging, in from ten to twelve hours after, it is believed, the poison had entered his body. Although, from an absence of suspicion at the time of the illness, the vomited matters were not examined, there can be no doubt that some arsenic must have been ejected with them. On an inspection of the body, Dr. Penny found *eighty-eight grains* of arsenic in the stomach and its contents, irrespective of a portion contained in the intestines and the quantity deposited in the viscera (Report of Trial, p. 51). Dr. Christison stated, in cross-examination, that he did not recollect any case of a person murdered by arsenic in whose stomach so much as eighty-eight grains had been found after death. At the same time he admitted that a large quantity might be administered in certain articles of food, *e. g.* cocoa; and that a much larger quantity must have been swallowed by deceased than was found in his stomach. This was made a turning point of the defence; it was contended that so large a dose could not have been taken unknowingly, and, therefore, that it indicated suicide. The learned counsel argued: "It is a dose which, so far as experience goes, never was successfully administered by a murderer. There is not a case on record in which it has ever been shown that a person administering poison to another, succeeded in persuading him to swallow such a quantity." It need hardly be remarked that persuasion has nothing to do with this inquiry. Could a man unknowingly take so large a dose, if secretly administered by another? Generally speaking, persons are destroyed by a much smaller quantity than was here found; but Dr. Christison, since the trial, has placed on record a case of *murder* by arsenic, in which from ninety to one hundred grains of this poison were found in the stomach after death! The person had survived from five to seven hours, and there had been frequent vomiting of a yellowish or greenish liquid during this period. The arsenic was administered in whiskey-punch with sugar, and it was kept in suspension by constant stirring (Edinburgh Monthly Medical Journal, December, 1857, p. 481, and Pharmaceutical Journal, January, 1858, p. 382). Had this case been brought forward at the trial of M. Smith, the facts would have neutralized a part of the defence on which the public were induced to place a great, but wholly unjustifiable, reliance. No medical jurist could admit that the discovery of eighty-eight grains (only half a teaspoonful) of arsenic into the stomach was inconsistent with an act of homicidal administration; and yet the defence rested in a great degree on this very slender point.

3. The *administration* of poison. A careful analysis may occasionally throw light on the question—who administered the poison? In the case of Humphreys (ante, p. 113) the discovery of sulphuric acid on the night-dress of the prisoner was an important fact to identify the woman as the person who had administered the poison. In the case of Wooler (ante, p. 108), arsenic was found in a syringe used for the purpose of injection. This proved not only how the poison had been administered to the deceased, but it had a tendency to fix the crime on a particular person. In Hartley's case (Central Criminal Court, May, 1850), the prisoner, a girl, was charged with attempting to administer oil of vitriol in coffee to her father. The prisoner usually made the coffee for breakfast, and would then have had an opportunity of adding it to the liquid.

It might, however, have been mixed with the coffee in the cup after it had been poured out; and, in this case, other persons had had the opportunity of poisoning the coffee. This question was solved by the aid of chemistry. I procured the coffee-pot, and found that it was old and rusty; the poisoned coffee was tested, and it contained no trace of iron, but on warming a small quantity of the acid coffee in the pot, it was immediately and strongly impregnated with sulphate of iron. It was, therefore, clear that the acid had not been mixed with the coffee in the pot, and might have afterwards been put into the cup without the knowledge of the prisoner. Numerous other cases will suggest themselves in which a guilty person may be detected, and an innocent person protected, by the aid of a chemical analysis of the poisoned food.

In the case of Reg. *v.* North (Guildford Summer Assizes, 1846), the proof of administration rested in part on the carbonizing action of oil of vitriol on sugar. Mary North was tried for the murder of an infant by giving to it oil of vitriol. The deceased had died from the effects of the poison; therefore the only part of the case which created difficulty, was the proof of administration. The mother of the deceased, wishing to give the child some aniseed-spirit and water, placed a lump of sugar in a white cup, and added a teaspoonful of the spirit; she then went to another apartment, and poured from a kettle about a teaspoonful of water. She observed no particular appearance in the mixture; she *tasted* it, there was no hot or acid taste, there was no blackening or change of color; she then gave the mixture to the infant, while a little girl who was present *drank up the dregs*, and suffered no ill effects. The prisoner was present, and in about half a minute took the child. After the child had taken this liquid, there were no symptoms or effects to attract attention, and it appeared relieved of the wind from which it had suffered. The mother left the room, and the prisoner then took the infant into an adjoining pantry, in which it was sworn there was a bottle of vitriol, put there by the prisoner. In about a minute and a half or two minutes, the mother, owing to a noise which she heard, returned to the room, and found her infant evidently writhing in great pain, and its mouth covered with a whitish froth. The prisoner, while bringing the child from the pantry into the kitchen, was, according to one witness, in the act of wiping its mouth with a napkin, on which sulphuric acid was found. Medical assistance was immediately sent for; but in spite of the best treatment, the child died. The defence was, that the mother had made a mistake, and given a teaspoonful of oil of vitriol in place of aniseed; but that was impossible under the circumstances, as the child who drank the dregs of the mixture did not suffer, and the mixture could not have been made as described by the mother, without blackening the sugar. In spite of these obvious conclusions, the jury acquitted the prisoner; but the medical facts of the case rendered it impossible that the mother could have made the mistake suggested. (See Guy's Hospital Reports, vol. iv, p. 396.)

Delicacy of analysis.—In conducting an analysis, the smallest possible quantity of a suspected liquid or solid should be used. If all were used at one operation, doubts might afterwards arise in the mind of the analyst, which it would be out of his power to remove. By care and ordinary precaution, a few grains will give results as satisfactory as those obtained from several ounces: and there is this additional advantage, that a portion is saved for the corroborative experiments of other analysts, or for correcting those which may have been previously performed. As a general rule, only one-half of the substance delivered for analysis should be examined. With respect to the minute quantities of poison which may be detected by chemical processes, some remarks will be made hereafter. It is, indeed, fortunate for the ends of justice, that the poisons which are commonly selected by criminals, may be discovered when existing in proportions so small as to excite wonder and incredulity in those who are not much acquainted with this department of science. The opinion of an experimentalist as to the *presence* of poison is never based upon the *quantity* actually found; for the results may be as infallible with a grain, or even the hundredth part of a grain of some substances, as with many ounces. All tests have a limit to their action; and when they act obscurely, or cease to act, a witness is bound to state that the chemical evidence has failed. Arsenic may be, however, safely inferred to be present when we obtain a quantity of the metal scarcely ponderable in the most delicate balance; but the crystals may be plainly visible under a good microscope. We might go on with the experiment, and obtain from other portions still larger quantities of the metal; but the evidence of the presence of the poison would not be, chemically speaking, rendered more conclusive. A toxicologist obtains sufficient to enable him to speak safely to the actual presence of the substance: what the weight or other physical properties of the quantity so obtained may be, is a matter of no moment to him. It is customary for some medical witnesses to say that they obtained only *feeble evidence* of a poison by the application of tests. The use of these terms is liable to give rise to an erroneous impression. Either there is chemical proof of the presence of poison, or there is not:—the law knows of no intermediate stage of evidence, nor will it accept it as proof; and a witness will assuredly expose himself to a severe cross-examination who makes use of this ambiguous language upon a question of such vital importance. The tests may reveal a very small quantity—but the results should not in any case be *doubtful;* they must be certain and decided, or they are worth nothing. The quantity of poison to which tho tests are applied, is left entirely to the judgment of the witness. It is necessary to observe in this place, that as the results of the action of tests depend in most cases upon the production of colored precipitates, no reliance can be placed upon experiments which are performed by *artificial* light. Clear and open daylight is indispensably necessary to the analyst. By artificial light white precipitates acquire a yellowish tint; yellow precipitates appear white; red, brown; and in some instances the action of a test is completely obscured. Counsel would be fully justified in objecting to any analysis based upon results obtained under these circumstances, where the opinion of the analyst is derived from a change of color.

Poisons in tests and apparatus.—If a practitioner has not been in the habit of analyzing poisons, it is advisable, before he commences an analysis of the substance handed to him, that he should operate several times upon a portion of the same kind of poison, as that which is suspected to have been administered. In the employment of chemical tests, it is especially necessary to determine that they are pure before an analysis is commenced. Arsenic may be contained in the sulphuric or muriatic acid (see Otto, Ausmittelung der Gifte, 1856, 105), or in the zinc used in the analysis of a substance suspected to contain this poison; and sulphuric acid may be pronounced to be present in the stomach when it may have been contained in the nitric acid employed in an analytical process. I have found antimony as an impurity in muriatic or hydrochloric acid, and lead as an impurity in acetic acid, to the amount of nearly two per cent. by weight. Solutions of potash or soda, or their carbonates, if kept in flint-glass bottles, speedily acquire an impregnation of lead, and a suspicion might thus arise that a poisonous salt of this metal was present in a liquid to which they had been added as tests. Carbonate of ammonia generally contains lead as an impurity, and I have also found traces of this metal in bicarbonate of soda and tartaric acid. In fact, all tests or reagents should be tested before they are employed for an analysis. The vessels used in these processes should be made of glass or of glazed white porcelain, the glaze having been previously tested by acids.

Danger of premature opinions.—During the examination of a suspected substance, an analyst is often pressed to give an opinion respecting its nature before the steps of the process are complete. This may arise from the anxiety or curiosity of those who are interested in the proceedings. There is a rule, however, which it appears to me should be always followed on these occasions: namely, that no opinion whatever should be expressed until the *whole* of the analysis is complete. It often happens in the hands of the ablest analyst, that the last steps of a process lead to a result very different from that which was anticipated at the commencement. The truth is, it is not by one character, but by many, that poison is identified; and, therefore, a suspicion derived from a few incipient experiments, is very likely to be overthrown by continuing the investigation. In the Boughton case, Dr. Rattray gave an opinion in the first instance, that the poison administered to the deceased was arsenic; but he subsequently attributed death to laurel-water. A case occurred, within my knowledge, in which arsenic was pronounced to be present when sulphuric acid was really the poison. In a case, tried at the Kingston Assizes, a medical witness admitted that, at the coroner's inquest, he stated the poison to be arsenic, but by subsequent experiments he found that it was oxalic acid. In another case which was the subject of a trial, the poison was at first stated to be oxalic acid, but on a more careful examination it was shown to be arsenic!

A mistake respecting the nature of a poison not merely impedes the course of justice, by throwing a doubt upon evidence which ought to be, beyond all question, clear and satisfactory, but it seriously affects the reputation of a witness. It generally arises from his giving an opinion before he is justified by the facts in so doing. It is, I think, a well-marked line of duty to be pursued on these occasions: 1. That no opinion should be formed from a *few* experiments; and 2. That no opinion should be expressed until the analysis is *completed.* It is obvious that if a man be compelled to admit in cross-examination at a trial for poisoning, that he has been once mistaken on a question so important, and requiring so decided an answer, a jury may be easily induced to believe that the witness may have made a second mistake, and that his then positive opinion is of no more value than that which he first expressed and afterwards retracted. (On the danger of trusting to an imperfect chemical analysis, see Annales d'Hygiene, 1829, ii, 405; xxvi, 399; xxix, 103, 474.)

Fallacies in chemical analysis.—The improved character of medical evidence is perhaps in no instance more strongly manifested, than in the facility and certainty with which, in careful hands, the chemical analysis of poisons is now conducted. A hundred years ago, chemical tests were either unknown, or so improperly applied, that really innocent persons, charged with the crime of poisoning, incurred great risk of their lives, in consequence of the chemical mistakes into which the medical witnesses of those days were so apt to fall. In some cases, although right in their inferences respecting death by poison, they were entirely wrong in their analysis. The evidence of Dr. Addington,—a most eminent physician of his day,—at the trial of Miss Blandy (Oxford Assizes, 1752), for the murder of her father by arsenic, furnishes a striking instance of a series of chemical errors. Dr. Paris has very properly observed, that not a single substance employed by the witness could be regarded as a test for arsenic; he inferred the presence of the poison from certain chemical decompositions which could not possibly be ascribed to it! The female charged with the crime was convicted and executed; but fortunately her conviction did not depend upon this extraordinary specimen of forensic chemistry.

In the chemical evidence given upon some modern trials, there has been one source of error against which a witness should sedulously guard himself. It is not sufficient to apply tests to show negatively that a suspected poison is contained in a substance requiring analysis, but its nature must be clearly demonstrated by a series of affirmative results. A case occurred a few years since, in which oxalic acid was *inferred* to have been the substance which destroyed life, because the liquid was acid, and no other acid poison could be detected in it! This kind of evidence can never amount to proof. It is better to express no opinion at all, than to base a conclusion on probabilities.

We are perhaps hardly yet acquainted with all the fallacies to which *individual* tests are exposed: the extension of chemical science is daily adding to their number by bringing out analogy of properties where it could not have been suspected to exist. The usual means of avoiding a difficulty of this kind is to employ *several* tests for the determination of the most important characters of a substance, and never to rely upon the action of *one* test only. It must be remembered, that if the poison be really present, not a single test ought to fail in its reaction except from circumstances which it would be easy to understand and explain. Under each poison the objections hitherto known as applicable to the tests will be stated, and the means of avoiding fallacy from their use, explained.

It was at one time believed that meconic acid was the only substance which gave a deep-red color on the addition of a persalt of iron, and the iron-test was thus supposed to indicate beyond doubt the presence of opium. Some years since, it was discovered that another acid (the sulphocyanic), which is naturally contained in the saliva, would produce the same effect; and precautions are now necessary to avoid the fallacy that may arise from a liquid containing saliva being supposed to contain opium. In a case mentioned by Orfila, MM. Ruspini and Cogrossi were completely deceived by the color-test for morphia (iodic acid and starch), on which, on the authority of Liebig and Serullas, much reliance was formerly placed. They found that a decoction of the viscera of a calf, which had taken no poison, gave the same reaction with the test, as the supposed poisoned articles which they were engaged in examining. I have heard of a case in which, from the application of the same test, morphia was supposed to be contained in, and eliminated with, the urine. It was subsequently found, that both lithic acid and the lithate of ammonia (constituents of healthy urine) produce the same change in iodic acid as morphia, and that this had given rise to the error. These remarks apply to strychnia and other alkaloids, as well as morphia. The color-tests are useful, when we can obtain an alkaloid crystallized in substance; but the mere indications of color, although they may give rise to suspicion, cannot be relied on as conclusive evidence. M. Devergie remarks (Méd. Légale, tom. iii, p. 17), that nothing is so deceitful as an absolute reliance upon color in testing. Four persons may look at the same colored product, and it will be found to present to each a different shade or tint. This especially applies to those cases in which the quantities are extremely minute. In spite of these well-known facts, however, there has been a disposition of late years to overrate the value of these tests, and to infer the presence of a poison from such a minute fractional proportion of a grain, that no corroborative process could by possibility be applied.

A reliance upon minute and uncorroborated results, led Orfila to affirm, erroneously as it has since been proved, that arsenic was a normal constituent of the animal body. A rash confidence in the tests for morphia, when applied to organic liquids, led an English "expert" recently to swear to the presence of this alkaloid in the stomach, and to its having been the cause of death, in a case in which he was subsequently obliged to admit that he was in error. (Pharmaceutical Journal, Jan. 1858, p. 350; and Feb. 1858, p. 433; also Guy's Hospital Reports, Oct. 1857, p. 497.)

When but infinitesimal traces of poison are discovered, and large quantities of materials have been used for its extraction, as in what Dr. Christison has properly designated the "enthusiastic" analyses of some French medical jurists, it would be unsafe to base any conclusion upon the results. Thus, in certain cases in France, the medical witnesses boiled up and evaporated the whole of the human body with many gallons of water and acids in large iron cauldrons, and inferred that the deceased had died from arsenic because they had detected in his remains infinitesimal traces (the 130th of a grain) of arsenic! The dramatic effect of these gigantic researches, was probably never more strikingly displayed than in the well-known case of Laffarge. The body of the husband was undergoing decoction in large iron vessels outside the court while the wife was on her trial for the murder within! The quantity of sulphuric acid, nitric acid, and nitre, which must be used on such occasions, is so great, that there is good reason to suspect the probable introduction of small traces of poison *ab extra*. A jury would, undoubtedly, be fully justified in rejecting chemical evidence procured by such means: and in any similar case the witness ought to be called upon to state whether he has previously examined for poison, *equal quantities* of the substances which he employed in the analysis. Evidence of a much less ambiguous character has been frequently rejected by a criminal Court in England.

The following incident, reported by Mr. Aickin in the Transactions of the Belfast Pathological Society, shows in what an insidious manner, poison may be unknowingly introduced into an article reserved for analysis. He was engaged in examining the body of a child, in order to determine the cause of death. The organs were healthy, and as no sufficient cause presented itself, he removed the stomach with a view of making an analysis of its contents. He was suddenly called away; and, to preserve the stomach, he wrapped it in a piece of paper (used for papering rooms), placing it on the uncolored side, and he locked it in a closet until the following day. Assisted by a friend, he then analyzed the contents, and found a trace of morphia with a pretty large quantity of arsenic. As the symptoms from which the child had died were not those of poisoning by arsenic, and there was no appearances of the action of arsenic on the body, he came to the conclusion that there must be some extraneous cause to acount for the presence of this poison. He examined a portion of the wall-paper in which the stomach had been wrapped, and then found that that part of it which was colored yellow, was tinted with sulphuret of arsenic or orpiment! It was therefore evident, as orpiment contains white arsenic, that the stomach and its contents had imbibed a portion of the poison during the night. (Lancet, June 23d, 1855, p. 632.) This satisfactorily accounted for the presence of arsenic under circumstances which might have given rise to a false charge of murder. Nearly all wall-papers, having any tinge of green or golden yellow in them, contain arsenic, and this arsenic spreads by imbibition to other parts of the paper not so tinted. It would, of course, be proper to avoid in all cases the use of any wrapper having upon it mineral colors of any description. Mr. Aickin's case shows in a striking point of view the danger of trusting to chemical analysis alone. Unless we look to physiology and pathology, a most erroneous opinion may be expressed.

According to Professor Otto, of Brunswick, arsenic is generally found mixed with iron in all ochreous deposits. Even in the fur deposited in tea-kettles, in which there is generally some oxide of iron, he found arsenic. From about a pound and a half of the crust or fur of a vessel, and from boiling water, he obtained well-marked arsenical deposits. Pöllnitz has detected in the fur of kettles, copper, lead, tin, and even antimony. Otto discovered a much larger proportion of arsenic in the calcareous crust taken from a kitchen boiler. Ten ounces of this gave a deposit of arsenic in a glass tube, and several stains on porcelain. He thinks that if a sufficient quantity be employed, arsenic will be found in all spring and well waters (Ausmittelung der Gifte, 1856, p. 61). These facts, if they prove anything, tend to show the extreme danger of placing reliance on minute chemical results in the absence of good physiological and pathological evidence (see ante, page 172). As hydrochloric acid may contain both arsenic and antimony, and this acid is largely used in the making of unfermented bread, these metallic substances may thus find their way into the body, and the presence of the metals be assigned to other sources. In May, 1856, a sample of this acid was sent to me from Bath, for examination. It had been used for making unfermented bread. Many persons who partook of this bread were seized with violent vomiting. I found the acid to contain a large quantity of chloride of antimony.

Hydrochloric acid is now so commonly sold contaminated with arsenic, that it is difficult to obtain it in a pure state. Some serious mistakes have occurred by reason of the use of arsenical acid in an analysis. In the case of Wooler (Durham, 1855), the acid employed in one set of experiments was found to be impregnated with arsenic. This discovery was not made until after it had been used for a medico-legal analysis. It is proper in all cases to test this acid, as well as the apparatus used, before any portion of a suspected substance is submitted to examination.

CHAPTER XII.

ON THE EVIDENCE OF POISONING FROM EXPERIMENTS ON ANIMALS—UNCERTAINTY OF RESULTS—CIRCUMSTANCES UNDER WHICH THIS EVIDENCE IS ADMISSIBLE—RECEIVED ON VARIOUS TRIALS FOR MURDER BY POISON—INJECTION-EXPERIMENTS—IS THE FLESH OF POISONED ANIMALS POISONOUS?—ANIMAL FOOD POISONOUS IN CERTAIN CASES—RENDERED POISONOUS BY CERTAIN VEGETABLES.

TOXICOLOGISTS have enumerated experiments upon animals as one among the sources of proof in cases of poisoning. This kind of evidence rests upon the assumption, that poisons act in the same manner on man and the lower animals. According to Orfila, this is partially true with only two domestic animals, namely, the dog and the cat: in other cases the results by no means accord. With respect to experiments performed on dogs and cats, I agree with the opinion expressed by M. Devergie (Médecine Légale, ii, 457), that they are in no case fitted to show the *doses* in which particular poisons are injurious or fatal to man—and they cannot be safely trusted to prove the rapidity of action of different poisons, or the rate of absorption, deposition, and elimination. All that they are fitted for, is to enable us to ascertain whether a particular substance be injurious to animal life or not; and, in some cases, its physiological operation, as well as the pathological changes produced by it. In Donellan's case, this kind of evidence was admitted to show the poisonous effects of laurel-water; and in Freeman's case, tried at Leicester in April, 1829, the results of experiments on animals were received as evidence to prove how speedily prussic acid, in certain doses, will destroy life. The experiments rather led to the presumption that the prisoner was guilty of the murder of a female by administering to her prussic acid; whereas, it was proved by circumstances that he was innocent. An exclusive reliance upon results so obtained, is always liable to lead to erroneous medical inferences. In some experiments made on dogs by Dr. Reid and Dr. Simpson they gave *an ounce* of Scheele's prussic acid to one animal; it died in about a minute afterwards. Other dogs of the same size, to which about *six drops* of the same acid, from the same bottle, were given, died in the same period of time; although the dose in the last case was only one-eightieth part of the quantity given in the first experiment. The contractility of the heart was in none of the cases much impaired. (Ed. Med. and Surg. Jour. Oct. 1836, p. 500.) From these and similar facts, it is evident that no correct inference can be drawn of the relative effects of prussic acid on man and animals; for there is no agreement as to the action of the poison on the latter. Doses so widely differing from each other, were found to kill dogs of similar size and strength within the same period of time.

When the question is merely, whether a suspected substance administered to another, is or is not poisonous, then we may occasionally be justified in resorting to this kind of evidence, in order to determine the fact,—particularly in reference to substances which have not been taken by and could not be safely administered to human beings. Most of the common poisons of the mineral kingdom are, however, capable of having their presence easily demonstrated by a chemical analysis; and the properties of the substance will be thereby known. But evidence of this description may be sometimes accidentally obtained, and then it may render unnecessary a chemical analysis of the vehicle of the poison; indeed, it may supply proof when no poison is discovered in the body of the deceased. The following is a type of many cases that have occurred in this country. A woman poisoned her husband with arsenic mixed in soup; and after the deceased had made a full meal, she threw the remainder out of a window into a farm-yard, thereby thinking to defeat all attempts at discovering the means which she had adopted to destroy her husband. It happened at the time, that a pig and several fowls were feeding under the window, and they ate up the food as it fell on the ground. The whole of these animals died under symptoms of irritant poisoning. The husband also died: no poison was detected in the stomach, although there were the traces of its action; but on opening the bodies of the animals, the medical witnesses found not only the appearances usually produced by irritant poisons, but arsenic itself was readily discovered in the viscera. This sort of evidence supplied that which was required to complete the case: for while no poison was detected in the body, no portion of the poisoned soup could be procured. The prisoner was convicted and executed.

Good negative as well as affirmative evidence may be sometimes obtained by the examination of the bodies of animals alleged to have been poisoned. In the case of Reg. v. Newton (Liverpool Autumn Assizes, 1856), it was proved that the prosecutor, to whom, as it was alleged, the prisoner had administered arsenic, went out into a back yard and vomited the food. Some fowls near the spot were observed to be ill during the day, and two died. The prisoner had in the meantime thrown away the poisoned food, and washed out the vessels which had contained it. As the prosecutor recovered, there could be no examination of his body, and a portion of the food which prisoner had prepared for herself contained no poison. Arsenic was, however, found in the crops of the chickens which had fed at the spot where prosecutor had vomited, and this supplied sufficient proof of the cause of his illness. A woman named Higgins was tried at the Warwick Summer Assizes, in August, 1831, for the murder of her uncle, by poisoning him with arsenic. Her guilt was throughout made very clear. It was proved that she had bought arsenic, and when required to account for the possession of the poison, she said that it was for the purpose of destroying vermin—the excuse resorted to by all murderers. She went, however, farther than this; and actually pointed out, in corroboration of her statement, a dead mouse, which she said had been killed by the poison. This turned out to be an unfortunate part of her defence, for the medical witnesses showed that the mouse had not died from the effects of arsenic.

In the above cases, it will be seen that the evidence from the effects of poison was accidental, and ancillary to the main facts of poisoning. There is, however, one instance wherein evidence from experiments on animals cautiously performed, may be of some importance on a criminal trial. I allude to the case in which a poisonous substance is not of a nature readily to admit of a chemical analysis, as for example in substances belonging to the neurotic class of poisons. In such a case, if the death of an animal takes place under the ordinary symptoms of poisoning from the administration of a substance, part of which has been taken by the person whose life was thus attempted, the evidence is conclusive. This remark applies to liquids or solids, which are made the vehicle of a poison, not to any matters vomited or found after death in the stomach. The results here would be fallacious; because such matters may, without containing any poison whatever, give rise to vomiting and other symptoms in an animal. The symptoms produced by some poisons, *e. g.* strychnia, are of such a special character, and the same in all mammalia, that a fair inference may be frequently drawn from the effects produced. Thus, in the case of Reg. v. Dove (York Autumn Assizes, 1856), the proof of the presence of strychnia in the stomach of deceased, was partly based on the effects produced on animals by a prepared extract of the contents. A sufficient quantity was procured to kill several animals under the usual tetanic symptoms produced by this poison. This evidence was conclusive, and more satisfactory than the application of chemical tests to extracts of organic matter containing the poison. Taking advantage of the extreme sensibility of the frog to the effects of minute doses of strychnia, the late Dr. Marshall Hall has proposed what has been termed the frog-test. A frog is prepared for the purpose, and placed in a bath containing a solution of strychnia, which may even amount to less than 1-100,000th (?) part of a grain. If the poison be present in this small proportion, the animal is suddenly seized with tetanus, and the body and limbs remain rigidly extended. If there be no strychnia the frog is unaffected. Taking this experiment alone, the result would not be sufficient to establish, beyond all doubt, the presence of strychnia, for these animals are readily tetanized by very slight causes, independently of strychnia; and without some corroboration the inference of the presence of this poison from such a result, would not, it appears to me, be justifiable. The negative evidence which it furnishes would, however, be better than the affirmative;

if a healthy frog had not symptoms of tetanus under the circumstances, it would go very far to prove the absence of any trace of that poison which would admit of detection by chemistry. The strychnoscopic test, therefore, must be regarded as an adjunct to other means of research, and not of itself sufficient to produce conviction.

When the food which is supposed to have caused symptoms of poisoning can be procured, this should be employed for the purpose of testing its poisonous or non-poisonous nature in preference to any liquid or solid removed from the body of the deceased. Foderé mentions a case, in which a child, after having partaken of some broth, fell into a state of stupor, lost all power of deglutition, and foamed at the mouth. Some of the meat from which the broth was made, was given to a cat. The animal was seized with convulsive fits, alternating with stupor, and died in about five hours. It was rendered probable from the symptoms, as well as from an examination of the body of this animal, that these effects were caused by the introduction of a narcotic plant (hyoscyamus) into the broth. (Méd. Lég. iv, 72.) A remarkable instance of this kind of evidence will be found under PHOSPHORUS, in which a shepherd and his dog were poisoned by this substance, which was detected in the stomachs of both after fourteen days' interment. In several instances this kind of evidence has been received in an English court of law. A woman named Sherrington was tried at the Liverpool Spring Assizes in 1838, for the attempt to administer poison to one Mary Byres. The evidence showed that the prisoner had sent to the prosecutrix a pudding, by two young children. On the way, these children tasted it, and finding that it had an unpleasant taste, the prosecutrix was put on her guard. The pudding was sent to a surgeon to be analyzed; but he could detect no poison in it. He suspected, however, that it contained a vegetable narcotic poison. He gave a piece about the size of an egg to a dog. In twenty minutes the dog became sick, in forty minutes it lost the use of its limbs, and died in three hours. The prisoner was convicted. Cases in which evidence of this kind, accidentally obtained, has been made available on charges of criminal poisoning, are now very numerous. (See Reg. v. Foster, Suffolk Lent Assizes, 1847.)

While experiments on animals may give us information on the nature of the symptoms produced by a particular poison, and on the power of discovering it in the absorbed or unabsorbed state in the body, they are not fitted to convey to us any accurate knowledge on the time of occurrence of symptoms, or on the dose required to cause death. In Freeman's case (ante, p. 195), the person accused narrowly escaped conviction and execution, by reason of undue confidence placed in experiments on animals, as evidence of the time of accession of insensibility in man, from a fatal dose of this poison. In that memorable case, which has furnished some point of illustration to almost every department of medical jurisprudence (Reg. *v.* William Palmer), an attempt was made to procure the acquittal of the criminal by straining the facts regarding the effects of strychnia on animals, and then applying them to the action of this poison on a human body. It was proved that no symptoms of poisoning by strychnia were manifested by the deceased, Cook, until an hour or an hour and a quarter had elapsed after he had taken certain pills, administered to him by the prisoner, which it was alleged contained the poison. According to the opinions of two of the medical witnesses retained for the defence, Mr. Nunneley of Leeds, and Dr. Letheby of the London Hospital, the symptoms could *not* be ascribed to strychnia, because the interval was too long! Their experience, they admitted, was chiefly derived from experiments on animals. Mr. Nunneley had experimented on sixty animals, including dogs, cats, rats, mice, guinea-pigs, rabbits, frogs, and toads; and the time of occurrence of symptoms from the ingestion of the poison was from two to thirty minutes, more generally about five or six. Dr. Letheby stated that he had seen "some dozens" of cases of the administration of strychnia to animals, and the average time when the symptoms began was a quarter of an hour. He had seen them begin in five minutes, and the longest interval was three-quarters of an hour. In answer to a question, he said, "I have never witnessed such a long interval between the administration of the poison and the coming on of the symptoms as in this case."

In an experiment with strychnia, performed by Dr. Geoghegan on a cat, no symptoms of poisoning appeared for *eight hours*. In another experiment, communicated to me by Mr. Holman, no symptoms appeared in a dog for *five hours;* and in a third instance, communicated by Mr. Ashwin, there were no symptoms in a dog for *eleven hours*. In an experiment by Dr. Christison, no symptoms appeared from a fatal dose given to a rabbit, for an hour and three-quarters; and in one out of five experiments by Dr. Rees and myself, no symptoms appeared for an hour and five minutes. It is perfectly clear, from these observations, that under the guidance of a counsel but little scrupulous as to his facts, or the sources whence they were derived, experiments on animals may be made to prove anything in a question of poisoning. Sergeant Shee, while trying to impress the jury with the notion that the accuracy of information was in a direct ratio to the number of animals slaughtered, was actually leading their minds to adopt a view which the most ordinary experience could have proved to be erroneous. For the first time, probably, on a trial for murder in this or any country, the counsel for the defence, in the case of Palmer, asked permission of the Court that the jury might be permitted to witness the poisoning of a few animals by strychnia, in order that they might draw their own inferences as to the nature and course of symptoms, and the period of death. This the Court very properly refused to permit. It is obvious that men in the position of the jury could not be in a condition to draw correct inferences from such experiments. They had already led into a gross error the "expert" witnesses called for the defence, and might have more readily deceived a jury composed of non-professional persons. It is not true that the symptoms caused by strychnia in man or animals are never delayed beyond three-quarters of an hour. The facts above related show that there is a considerable variation in the length of the interval among animals; and in a human being, the time for their occurrence, which was the subject of dispute at this trial, has been actually exceeded in several instances of poisoning by strychnia (see ante, p. 114).

Is the flesh of poisoned animals poisonous?—This is a question which it is necessary to consider, because poultry and game are not unfrequently poisoned wilfully or accidentally, and in this state they may be eaten unsuspectingly. It is well known that grain is often saturated with a solution of arsenic for agricultural purposes before it is sown: if this grain be eaten by poultry, it will destroy them; and a question may arise as to the effects which the flesh of the animals so poisoned, is liable to produce on man. In other instances poison has been placed in the way of these animals, with the malicious object of destroying them. Thus wheat or oats saturated with arsenic, or with that poison intermixed, have been placed in game preserves, for the purpose of destroying pheasants and other birds. In the spring of 1846, two blackcocks were sent to me for examination, from the extensive preserves of a nobleman in Scotland. They had been found dead on the grounds. A quantity of arsenic was discovered intermixed with oats and the shoots of the larch, in the crops and gizzards of each bird, and arsenic existed also in the pectoral muscles and soft organs. There had been previously a very large destruction of game on the estate, as it was inferred, from poison. Many similar cases, in which poultry and game were poisoned, have since come before me. There is hardly a doubt that, when the animal dies soon after the ingestion of poison, and obviously from its effects, the flesh would be poisonous to man, although it might require a large quantity of the flesh to produce a fatal result. Dr. Christison reports a case which renders this opinion highly probable. (On Poisons, 81.) This subject has been examined in reference to sheep by M. A. Guérard. (Ann. d'Hyg. 1843, i, 468.) Some sheep were poisoned by arsenic, and it became important to determine how far their flesh was rendered poisonous as food. A Commission was appointed by the French Academy to make inquiry respecting the facts, and Mr. Guérard has furnished a summary of the results. The sheep submitted to experiment appeared well, even when they were daily passing arsenic through the fæces and urine. On giving to a young dog the flesh of a sheep which had died from arsenic, the animal after two days was seized with diarrhœa, and arsenic was detected in the fæces and urine. Another dog, which ate the viscera, previously washed, had vomiting and symptoms of a more serious kind; it became thinner, but did not die from the effects of the poison. These results prove that the flesh of poisoned animals is noxious; but if they live sufficiently long, the whole of the arsenic is voided in the urine and fæces, and the flesh may afterwards be eaten with impunity. In an experiment on one sheep, arsenic was found in the fæces twenty-two hours after the introduction of the poison into the stomach. Its elimination was daily traced; and fifteen days after it had ceased to appear in the fæces, it was found in the urine. It ceased to appear in the urine on the thirty-fifth day: and when the animal was killed on the thirty-eighth day, no arsenic was found in its body. Six persons ate of the flesh without suffering any ill effects, and a dog ate the viscera without manifesting any symptoms of poisoning. The flesh, therefore, is only noxious in the early or acute stage of poisoning, and it is not fit for food until three or four days after arsenic has ceased to appear in the urine. Arsenic and corrosive sublimate are much used in this country as a lotion for the purpose of destroying the fly in sheep; but it is not likely that any question will ever arise respecting a poisonous impregnation of the flesh from this source, unless the animals be speedily killed. In a case reported by Mr. Annan, two sheep died from the effects of the external application of corrosive sublimate, a poison which is most easily absorbed. (Med. Times, July 25, 1846, 331.) The flesh of these animals might have proved noxious if it had been eaten.

The flesh of animals poisoned by copper has been known to produce serious effects among those who have eaten it as food. Dr. Galtier relates the following case. A pig which had been fed with corn soaked in blue vitriol, was so affected that the owner had it killed, and sold the carcase to a butcher. Seventeen persons, who ate of the flesh of this animal, were seized with violent colic, and those who ate the blood made into black puddings, also suffered severely. The milk of a goat which had eaten sour food out of a copper vessel, occasioned nausea, vomiting, colic, purging, cramp, and other alarming symptoms, among fifteen persons who partook of it. They had before taken milk from the same goat, without injurious consequences. The animal itself became ill, and died on the third day, under all the symptoms of poisoning. The mucous membrane of the small intestines was found inflamed. (Toxicologie, i, 631.) It is to be regretted that no analysis of the food was made in these cases of acute poisoning.

But little is known concerning the effect produced by absorbed organic poisons on the flesh of animals. It might be supposed that a few experiments with such a poison as strychnia would at once supply an answer to the inquiry. I have frequently seen the larvæ of insects grow and thrive on the flesh of animals killed by strychnia; but then, on experiment, I found that the larvæ were not killed by the application of a strong solution of sulphate of strychnia to their bodies, and they did not appear to be in any way affected by a dose of the poison which would have speedily destroyed a rabbit. Dr. Harley found that the flesh of animals killed by minimum doses of strychnia, did not act as a poison to other animals. He fed a hedgehog on poisoned flesh for a period of fourteen days, without being able to detect the slightest symptom of poisoning. The poison must, therefore, he concludes, have been either decomposed, or it was not present in sufficient quantity. (Physiological Action of Strychnia, p. 15.)

Dr. Macadam states that he killed a horse with thirty-two grains of strychnia, given at intervals. He fed a large-sized terrier dog for two weeks on the flesh of this horse; the animal eating every day during this period two pounds of muscle. The terrier dog lived and throve on the flesh, and did not betray the faintest shadow of tetanic symptoms. He states that, on analysis, he found distinct evidence of strychnia in the muscle and blood of the horse. (Pharmaceutical Journal, August, 1856, p. 124.)

Bernard gave, as food, to dogs, rabbits which had been poisoned by strychnia and nicotina, and the dogs experienced no ill effects. When, however, a larger dose of poison, than was sufficient to cause death, had been given, then the surplus acted fatally on other animals. This would be, in fact, a case of direct poisoning. But the flesh alone does not appear to have any poisonous action, the quantity of absorbed poison contained in it being too small. The intestines of animals poisoned by strychnia and nicotina, have proved fatal to other animals (Léçons sur les Effets des Substances Toxiques, 1857, p. 281). From these observations, therefore, it appears that it is not the absorbed, but the unabsorbed portion of the alkaloidal poison, which is likely to affect other animals.

The results obtained by Drs. Harley and Macadam, as well as by M. Bernard, bear out the view that the flesh of game killed by the arrows of certain Indian tribes in South America, poisoned with woorara, may be eaten with impunity. The quantity actually absorbed and diffused through the flesh is, as in the case of strychnia, too small to cause symptoms of poisoning, or the organic poison undergoes some change in the body by which its noxious effects are destroyed.

It is a curious fact, that the bodies of animals may, in some instances, be made the vehicles of transferring poison to the human subjects, while the animals themselves do not suffer from its effects. Thus the flesh of the pheasant, which feeds on the buds of the *Calmia latifolia*, in North America, is deemed poisonous during the winter and spring. (Beck's Med. Jur. 854.) The flesh of hares which have fed upon *Rhododendron chrysanthemum*, is considered to be poisonous. A singular case occurred in France, in which a whole family near Toulouse was poisoned by a dish of snails. The symptoms under which they suffered were those of narcotico-irritant poisoning; and it was found upon inquiry, that the snails had been gathered from bushes where they had fed upon the leaves and young shoots of the *Cariaria myrtifolia*, a vegetable poison. (Gaz. Médicale, Oct. 1842; also Med. Gaz. xxxi, 237.) It has been long known that honey, derived from bees which have fed upon the *rhododendron calmia*, *azalea*, or *datura*, growing in certain districts, acts as a narcotico-irritant poison, producing vomiting, purging, and giddiness. In the chapter on ANIMAL IRRITANTS some other facts will be mentioned, from which it would appear that the milk of cows fed in certain districts of America is poisonous, and gives rise to serious symptoms, whether taken as milk or made into cheese. The flesh of the animal possesses also poisonous properties; while the animal itself does not suffer in health from feeding on the plants. These facts are explicable on the supposition that there are specific idiosyncrasies among different classes of beings, thus rendering what is innoxious to one a poison to another.

I have elsewhere (p. 198, ante) objected to certain physiological experiments on animals, as being liable to lead to incorrect results. All experiments based on the injection of poisons into the blood, are open to the objection that great violence is done to the circulation; and it appears to me that no inferences derived from them can be fairly applied to legal medicine. Any substance thrown into the circulation may thus destroy life by a mechanical action. M. Dupuy announced some years since that the *brain* of the sheep killed animals even more rapidly than *corrosive sublimate!* This extraordinary conclusion was derived from an experiment in which a solution of cerebral matter was injected into the crural vein of an animal, and death took place in a few minutes.

(Med. Gaz. xxxix, 745.) It is not at all improbable that death was caused under the circumstances, but it is preposterous to compare the effects of sheep's brain with those of corrosive sublimate. Upon similar crude experiments a solution of fæces, or of some most common articles of food, might be inferred to be more poisonous than arsenic!

CHAPTER XIII.

WAS THE DEATH OF A PERSON PRODUCED BY POISON, OR BY ANY OTHER LATENT OR SECONDARY CAUSE?—DEATH FROM UNWHOLESOME FOOD—DEATH FROM DISEASE SIMULATING POISONING—POISON FOUND IN THE BODY NOT A CAUSE OF DEATH—CASES OF SUSPECTED POISONING—DEATH FROM VIOLENCE SUPERVENING ON POISON—QUESTIONS OF HOMICIDE AND SUICIDE—OF TWO POISONOUS SUBSTANCES TAKEN BY THE DECEASED, WHICH CAUSED DEATH?—DEATH FROM POISON, AND NO POISON FOUND IN THE BODY.

We have hitherto considered those facts which indicate in a disputed case whether poison has or has not been the *cause of death*, in a previously healthy subject. We have supposed that the question of poisoning would turn simply on the affirmative or negative, and be established or disproved by the medical evidence. We meet with cases, however, in medico-legal practice, wherein the question presents itself under another aspect. Thus poison may have been taken or administered; the fact of poisoning may be established by the symptoms, the appearances after death, and the actual discovery of the substance in the food, in the vomited matters, or in the stomach of the deceased. All these points may be freely conceded; but the defence will rest upon the question, "Whether or not, the poison so administered, was actually the cause of death?" To establish a charge of murder against a prisoner, it must be proved that poison was certainly and indisputably the cause of death. Any proof short of this, as the existence of mere probability, doubt, or suspicion, will of course lead to an acquittal. (See the case of Pouchon, Ann. d'Hyg. 1844, i, 431.) Thus, then, the medico-legal question would be: *Was death produced by poison, or by any other latent or secondary cause?* The witness will be required to state which of the two probable coexisting causes actually destroyed life. It may be remarked, that whenever we obtain those proofs of poisoning which have here been assumed to exist, the presumption is always in favor of death from poison; but it is not the less necessary for a medical jurist to determine, by a careful inspection of all the cavities of the body, whether death might not have been due to some insidious disease. In a case at all involved in doubt, negative evidence is as important as that which is affirmative; and a great error would be in many cases committed, if the examination of a body were stopped as soon as traces of the action of poison had been discovered. In Donellan's case, the head of the deceased, Sir T. Boughton, was not examined, an omission which, had the general evidence been less clear, might have led to difficulty; for the diseases from which it was afterwards alleged the symptoms of the deceased might have proceeded (apoplexy and epilepsy) have their seat of morbid changes in that part of the body. An inspection of the head might, it is true, have thrown no light upon the question; but this is not the point—a medical witness must not omit this duty, and then excuse himself by saying that no morbid changes might have been found. The assumption will always be as much against him as in his favor. Cases in which the administration of poison is admitted, and death is referred to some other cause, although not common in courts of law, are sufficiently frequent to demand the serious attention of the practitioner. The following appear to me to embrace the chief points on which a defence of this kind may rest.

1. DEATH MAY BE CAUSED BY IMPROPER FOOD.—It has been mentioned in a preceding chapter (ante, p. 101), that some kinds of food will cause death under symptoms resembling those of irritant poisoning. Such cases are not common, and they appear to depend often on idiosyncrasy, or peculiarity of constitution. If poison be taken with such food, we may safely refer death to the former, provided the case took the usual course; and that death was preceded by all or a majority of the symptoms peculiar to the kind of poison taken. If any of these characters are wanting, this must weaken the evidence; but in most instances, it will be found that the symptoms of acute poisoning are so well marked, as to extinguish those which may have depended upon unwholesome food. Each case must be judged of by itself; no general rules for a decision can be laid down. Still, it must be remembered, that death is not a very common consequence of unwholesome food, while it is the usual result of an active poison.

2. POISON MAY BE FOUND IN THE BODY, AND DEATH MAY BE CAUSED BY DISEASE.—This is a case which more frequently presents itself for our consideration; since poison is often wilfully or accidentally administered to persons while laboring under disease. On an examination of the body, we may find, besides indications of poison, marks of extensive disease. When this happens, the chief point to be considered is, whether the disease had advanced to that degree to account for *rapid or sudden death;* for this is one of the essential characters of acute poisoning. Should the history of the case be known, our judgment may be assisted by observing whether the symptoms preceding death were referable to a diseased condition of the body, or to poison. We cannot deny that singular coincidences may occur. A man may have taken irritant poison, and yet death be occasioned by abscess in the brain, or the lungs, by sudden hemorrhage, or other causes. If the poison were of a nature to cut short life suddenly, we could not hesitate to refer death to it. Thus it is scarcely possible to admit, when prussic acid is the poison, that death should be referred to some diseased condition of the body found on an examination of the body. Whether the person is laboring under illness or not, the taking of this poison would be sufficient to account for death. The only exception would be where the prussic acid

was in small quantity, and might have been derived from some accidental source. It is not always so easy, however, to determine this question in other cases of poisoning; for, whether the substance taken be opium or arsenic, there is time for latent disease of the heart, brain, or lungs, to cut short life. The history of the symptoms preceding death will enable us in general to return an answer. Without this history, or some strong corroborative evidence, a medical opinion can be little more than a conjecture.

Several complex cases of this description have occurred in reference to diseases of the stomach, the persons laboring under such diseases having had poison administered to them. Thus, the organ has been found perforated, and the question has been, not so much what caused the perforation, as whether the perforation or the poison had caused death. A woman swallowed, by mistake, half an ounce of powdered chloride of barium dissolved in warm water. Nausea and vomiting of a watery mucus supervened, with twitchings of the facial muscles, and convulsive motions of the hands and feet. The symptoms continued to increase in severity, and she died in about two hours from the time of taking the poison, under the most violent convulsions. On inspection, the stomach was found perforated posteriorly, in the lesser curvature, near the cardiac orifice. The aperture was of an oval form, three lines in diameter externally, and almost twice as large internally. The margin appeared swollen, and the mucous membrane for about two inches around was much thickened, and covered with a bloody mucus. The stomach and small intestines were highly inflamed; the cavity of the former contained mucus and coagulated blood. The pharynx and œsophagus presented slight marks of inflammation. The poison was found in the stomach by chemical analysis. Wildberg, who has reported this case, suggested that the perforation was due to the previous disease, and not to the poison taken. This is very probable, for the characters of the aperture were those of perforation from disease (ante, p. 159); and it would be very unlikely that the chloride of barium, if it led to perforation of the stomach at all, should have given rise to this effect in two hours. It is not stated whether the woman suffered from any symptoms of gastric irritation prior to taking the poison, nor whether the contents of the stomach were found extravasated and the peritoneum inflamed. But there can be no doubt that the woman died from the effects of the poison. This was clearly indicated by the nature of the symptoms, and the appearances after death. Admitting that no mistake was made respecting the time at which the poison was swallowed, it must be considered remarkable that this substance should have destroyed life, and left such extensive marks of irritation in the alimentary canal, in the short space of two hours.

The following case was tried at the Taunton Spring Assizes, 1836. A woman, named Edney, was charged with the murder of her husband, by poisoning him with arsenic. It appeared in evidence, that the deceased was attacked with severe pain in the abdomen and vomiting, shortly after having eaten his dinner, which was prepared for him by the prisoner. Medical assistance was called in; but the man became worse, and he died in sixty hours after the first attack. It was shown that arsenic had been probably given to him at the dinner; and also on several other occasions, when it was supposed to have been substituted for some medicine prescribed for him, his symptoms having been uniformly aggravated after each dose. The chemical evidence was very clear: arsenic was discovered in the vessel in which the dinner was dressed; also in the stomach of the deceased; and the poison was traced to the possession of the prisoner. On an examination of the body, a scirrhous ulcer, evidently of long standing, was found in the stomach, near the pyloric orifice. It was about the size of a shilling, had a dark appearance, and the margin was inflamed. The mucous membrane of the stomach, as well as of the duodenum, was in such a high state of inflammation that it resembled red velvet. The defence on the trial was, that the symptoms and death of the deceased were due to the scirrhous ulcer, and not to poison. It was shown that the deceased had suffered from a gnawing pain of the stomach for a very long period; and it was thought by himself, as well as by others who saw him, that this last attack of illness was nothing more than an aggravation of his old complaint. The medical witness, however, did not hesitate to refer the symptoms and death to arsenic, for the following reasons: The symptoms occurred suddenly and violently, after a meal, at which arsenic was proved to have been administered. Some of these symptoms were peculiar to arsenic, and totally unconnected with the disease under which the deceased was laboring. Pain and vomiting might be ascribed to either cause; but the intense thirst not previously experienced, well-marked inflammation of the eyes, coldness of the body, and, before death, paralysis of the extremities, with loss of sight, were symptoms unquestionably owing to the operation of arsenic, and were not the effect of chronic disease. This disease was not likely to destroy life with such rapidity and under such severe symptoms. The appearances corroborated the opinion founded on the symptoms, and showed that death was really due to an active and irritant poison. The woman was convicted upon this evidence.

The case of Reg. v. Foster (Suffolk Lent Assizes, 1847,) also furnishes an important caution to medical witnesses. The prisoner was here charged with poisoning her husband. He had died somewhat suddenly: his body was inspected, and death was referred, by the inspectors, to an inflamed state of the kidney, and a rupture, to a very small extent, of the inferior vena cava. The stomach and intestines were subsequently examined by Mr. Image and Mr. Newham, of Bury St. Edmunds, and a large quantity of arsenic was found in them. The whole of the tissues were completely impregnated with it. They properly ascribed death to arsenic, considering that the condition of the kidney was not a sufficient cause. The injury to the vena cava was no more than what might have arisen from an accidental puncture during the examination of the body. This case is otherwise remarkable from the fact that the prisoner was convicted, although there was no apparent motive for the crime, although no arsenic had been traced to her possession, and there was no direct proof of administration.

The next case occurred in Germany. A woman, after an illness of many weeks, during which she was subject to constant vomiting and other symptoms of disease in the stomach, died suddenly under suspicious circumstances, and her husband was accused of having poisoned her. The parties had lived unhappily together. The prisoner, under the pretence of relieving her disorder, gave her a white powder and a mixture of *Boletus cervinus*. Soon after taking this powder she became much worse, and severe pain in the abdomen with purging came on. She died nine days after taking the powder; and a physician who saw her shortly before death, considered her to be laboring under the effects of irritant poison. The deceased gave to the physician the glass from which she had taken the alleged medicine. This contained a white powder, which, on examination, proved to be arsenic. There were well-marked appearances of poisoning in the stomach. The whole of the interior was of a dull reddish-brown color: the lining membrane being in some parts so softened as to have a gelatinous consistency. About half an inch from the pylorus, there was a perforation of the coats of the organ. The edges of the aperture were hard, and had a cicatrized appearance. The stomach contained about twelve ounces of a reddish-colored liquid. The intestines were slightly inflamed. The medicine alleged to have been given by the prisoner to the deceased, was proved to have been arsenic in a decoction of the *Boletus cervinus*. The contents of the stomach and intestines yielded no trace of poison, but the analysis does not appear, from the report, to have been skilfully conducted. The medical opinion, given at the trial, was, that the deceased had died from arsenic, and not, as it was alleged, from disease. The prisoner was acquitted of the charge, the court doubting the correctness of the medical opinion in favor of death from poison. The witnesses were asked, whether they would swear, from the appearances *alone*, that the deceased had died from arsenic; but this they declined doing. The previous disease of the stomach, and illness of the deceased, were, in the judgment of the court, a sufficient cause of the symptoms and death. It is proper to state also, that the evidence of administration by the prisoner was defective. (Rust's Magazine, 1837, 50 B. 2 H.)

I have already referred to a case, in which a woman laboring under malformation of the heart was supposed to have taken nux vomica, and to have died from its effects. The facts, however, showed that death had been really caused by an obstruction to the circulation through the heart (ante, p. 146). The two following cases are related by Henke. A girl died under suspicious circumstances, and an inspection of the body was ordered. The viscera were found healthy, except those of the abdomen. The stomach contained three ounces of a reddish-colored liquid. Its mucous membrane was of a dark red color, and near the intestinal opening were several spots of a clear yellow hue. The contents of the stomach, on analysis, yielded arsenic. The account given by the mother was, that the deceased, some weeks before, had met with a fall, after which she complained of pain in her side. Shortly before her death she said she felt ill, and vomited repeatedly,—she went to bed early, and died without being convulsed. The medical opinion was to the effect that she had been poisoned; but the court held that the fact of poisoning was not proved, and the person who was charged with the crime was acquitted. This case shows that there is great difficulty in forming a medical opinion, when there is no satisfactory account of the symptoms preceding death. In the case of The Queen v. Jennings (Berks Lent Assizes, 1845), the deceased, although ill, was not seen by a medical man during life, but the stomach was inflamed, and arsenic was found in it. The only account of the symptoms preceding death, was derived from the evidence of a girl. The court held that the fact of poisoning was established.

In another case, a man was charged with having given to his wife, who had been for a long time out of health, a small quantity of arsenic in four different doses. The only symptoms that followed, were general illness and vomiting. Another and stronger dose was then, it is supposed, administered; and after suffering severe pain, the woman died the day following. The body was inspected twenty-four hours afterwards. In the abdomen, the pancreas was found enlarged and in a scirrhous state, evidently proceeding from chronic disease. The lining membrane of the stomach was inflamed, and it presented gangrenous spots. It contained a grayish colored liquid, having a gritty feel. The uterus was in a state of scirrhous enlargement. The contents of the stomach, on analysis, were found to contain arsenic. The medical opinion was, that notwithstanding the marks of extensive disease in the viscera of the abdomen,—the appearances after death, and the detection of the poison in the viscera, proved that the deceased had died from arsenic. The counsel, in defence, raised objections to this view, on the ground that the head had not been examined, and that the chemical analysis was defective. The Faculty of Leipsic being appealed to, overruled the objections. The diseased state of the pancreas might, in their opinion, have given rise to vomiting, emaciation, and death, but not to so sudden a death. The chemical analysis, although in some respects defective, sufficiently demonstrated the presence of arsenic in the viscera (Zeitschrift der S. A).

The following case is reported by Dr. Carson. (Monthly Journal of Medical Science, Aug. 1846, p. 142.) A sailor after making a hearty meal was seized with intense pain in the abdomen, and died, without any relief from treatment, in about fifteen hours. On inspection, a large opening was found in the stomach, and the whole of the mucous membrane at the cardiac end was intensely injected. The small intestines, from the stomach to the cæcum, were equally injected, and appeared like red velvet. A number of round worms (L. teres) were found in the small intestines, some of them still living. The other organs of the chest and abdomen were healthy. There had been an effusion of fluid into the cavity of the abdomen (through the perforation), but there was no sign of inflammation. Dr. Brett examined the stomach, its contents, and the fluids of the abdomen; and a trace of arsenic was discovered, so slight that Dr. Brett did not consider it sufficient to enable him to give an opinion that arsenic was the cause of death. The jury returned a verdict, leaving the real cause of death in doubt, *i. e.* that the deceased had died from inflammation and perforation of the stomach, which might have been produced by arsenic or natural causes. Dr. Carson considered that the rupture of the stomach was due to the rapid development of gas as a consequence of the acetous fermentation of the contents. As arsenic was really discovered in this case, it is obvious that it must have been taken by the deceased, although this does not appear from the symptoms and moral circumstances. What was the cause of death—disease or poison? Arsenic does not commonly produce perforation, and it is very unlikely that it should have caused a large aperture in the stomach in fifteen hours. But arsenic would account for the highly inflamed condition of the alimentary canal, and the actual quantity found in the body after death, however small, is of course merely a residue of what has killed. The report states that no effusion of lymph, or other sign of peritonitis, was observed. The perforation, therefore, if the cause of death, does not appear to have operated fatally in the usual way. The aperture in the stomach could not be ascribed to the worms, as these were found in the small intestines. This is a most difficult case on which to express an opinion, but, taking the whole of the facts, with the positive evidence of the presence of arsenic, it appears highly probable that death was caused by poison. The only other view of the matter is, that there was rupture of the stomach operating fatally by shock; but this leaves unexplained the extensively inflamed condition of the stomach and intestines.

In the following number of the same journal (Sept. 1846, p. 184), is another case by Dr. Paterson, in which no doubt existed that poison had been taken, but the cause of death appeared somewhat obscure. A girl, æt. 18, swallowed a drachm of King's yellow (a mixture of sulphuret of arsenic, lime, and sulphur). In about two hours she began to vomit, and she still vomited when admitted into the Infirmary, *i. e.* about ten or twelve hours after having taken the poison. When admitted, there was great anxiety, with collapse, coldness of surface, and a scarcely perceptible pulse. On the day following, the signs of irritation in the alimentary canal abated under treatment, and symptoms of acute bronchitis supervened. She died sixty hours after having taken the poison. Appearances indicative of inflammation were found in the air-passages and lungs: but there was no sign of active inflammation about the stomach, and the intestines were healthy throughout. There could be no doubt that death was immediately caused by bronchitis; but the question to be decided was, whether this had arisen from the usual accidental causes, or whether it had been produced by poison. From the fact that the mucous membrane of the air-passages has been occasionally found inflamed in cases of arsenical poisoning, Dr. Paterson concluded that the arsenic was here the remote cause, and that the inflammatory action probably extended by continuity from the alimentary canal into the air-passages. This question is of considerable importance. Let us suppose that some person had been charged with criminally administering the poison. Could he have been convicted of murder under the circumstances? Could a medical witness have sworn without hesitation that the poison produced the fatal bronchitis, and caused death; and that it was impossible that the disease could have arisen by any coincidence from accidental causes? It appears to me that such an opinion would not have been justifiable, because acute laryngitis, although it may by possibility occur, cannot be reckoned among the ordinary fatal consequences of arsenical poisoning; and it would be exceedingly difficult to produce a single instance in which arsenic had clearly and indisputably caused death by exciting inflammation of the mucous membrane of the air-passages.

When poison is found in a dead body, it is natural to refer death to this as a sole and sufficient cause; but it is the duty of a medical jurist to remember that there may be other causes of death in the form of latent disease. The case of Reg. v. Dore and Spry (Central Criminal Court, August, 1848) (Medical Gazette, vol. 42, p. 888, 1848) is in this respect worthy of notice. The accused persons (the mother and grandmother of the deceased child) were charged with murder by administering to it arsenic. The child at four months had suffered from dentition. On the 13th of July, it was suddenly seized with pain in the bowels, which was relieved by fomentations. The pain increased during the day, there was vomiting of bile, but no purging; some blood was passed once from the bowels. The medical man who was called in pronounced it to be a case of intussusception of the bowels (ante, p. 126), and gave no hope of its recovery. On the following day, the 14th, the child became progressively worse; the abdomen was swollen, and the usual remedies procured no relief to the bowels. Four grains of calomel were prescribed; the powder was mixed with a little milk, and given by the medical man in attendance to the infant while lying in a hopeless state. There was neither vomiting nor purging. The child died on the 15th July, about seven hours after the powder had been put into the mouth, and fifty hours from the time at which it was first seized with pain in the abdomen. On an inspection of the body five days after death, there was found an intussusception of the lower part of the small intestines, about one foot of the intestine being completely locked in another portion near the cæcum. The strangled part was highly inflamed downwards from this point, but *not upwards*. The duodenum and all the small intestines above the strangulated part were free from inflammation, and filled with feculent matter of a bright yellow color. The disease had gone on to that degree that the passage through the intestine was completely obstructed. This was considered to be a sufficient cause of death, and was certified accordingly. At the subsequent inquiry the evidence of the medical witness who made the inspection was to the effect, that the disease was in such an advanced stage as to be incurable, and that it would account for all the symptoms and for the death of the child. The stomach was not opened until a week after death; it was not inflamed, but there was a dark patch in one part fringed with a yellowish substance. This on analysis proved to be arsenic, calculated to amount from two to four grains. The dark patch was referred by the chemist who made the analysis, to gangrene produced by the poison. No trace of calomel or any mercurial preparation was found in the stomach; and through unpardonable neglect in a case of such grave importance, the chemist made no analysis either of the intestines, of their contents, or of the liver or tissues. He deposed in the most positive terms, that the arsenic must have been in the child's body *at least two days*, and that the poison could not possibly have been given twenty-four hours before death. On this chemical evidence the accused, who had had the custody of the child from the commencement of its illness, were put upon their trial for murder. No motive was apparent or even suggested, as the parties were in a respectable sphere of life. The main question turned on the cause of death, and the accuracy of the chemical evidence as to the date of reception of arsenic into the body of the child. Having been called as a witness on the part of the Crown, my evidence was to the effect that, taking the symptoms and appearances, they were not those of poisoning by arsenic, but of intussusception or obstructed bowels; that such a disease was a frequent attendant on dentition in children, and when existing to the extent described in this case, it would be likely to prove fatal. Having, under an order of the Court, examined the stomach, I gave my opinion that the dark patch ascribed by the chemist to gangrene, arose from altered blood effused as a result of the local irritant action of arsenic,—that the absence of inflammation from the stomach, duodenum, and small intestines, as far down as the strangled portion, rendered it in the highest degree improbable that the arsenic could have been in the body of this child two days, or even many hours; that so large a quantity of arsenic as from two to four grains could not have been in the stomach of an infant two whole days without producing urgent and violent symptoms, incessant vomiting and purging, collapse, and a more rapid death. The death of the child, therefore, in my judgment, was owing to intussusception, of which dentition was a sufficient cause. Had the arsenic produced the intussusception, there would have been clear symptoms of arsenical poisoning preceding the attack, attended by inflammation of the stomach, and probably inflammation of the bowels between the stomach and the seat of strangulation. That an infant of four months should live two days with a quantity of arsenic in its stomach sufficient to kill two adults, without producing inflammation of the stomach and bowels, would be contrary to the usual course of experience on the action of this poison. The theory of gangrene, on which alone the assumed date of administration rested, was, therefore, quite inadmissible. But it was necessary to account for the arsenic in the stomach. On minute inquiry, it turned out that the five grains of calomel given by the medical man to the child some hours before its death, were dispensed at night by a person in a shop, who admitted that it was not his duty to dispense medicine, and that white arsenic was kept in a bottle in the shop over the shelf on which the calomel was kept. This witness stated that he was "in a hurry" when he dispensed this powder, and gave the following evidence in cross-examination:

"The powder was white. I knew that white arsenic was kept in the shop, but I did not sell any that night. *By the Court:* I should not know calomel from arsenic by its appearance! I don't know whether arsenic is rougher in appearance than calomel. I never examined them together. I cannot say whether calomel, being mixed with sugar, would give it still more the appearance of arsenic."

As no calomel or mercurial preparation was found by analysis in the stomach of the child, and if given in so large a dose within a few hours of death, some portion must have been found, it appears only a reasonable presumption that a mistake was here made by an ignorant dispenser, and that arsenic was supplied instead of calomel. The recent introduction of the arsenic would account for the absence of symptoms and appearances; and in no other way could the absence of calomel be explained. There was nothing to show that the poison had accelerated death, for the child was at the time fast sinking from the fatal disease. Upon this evidence, Platt, B., who tried the case, addressed the jury, and they, without calling upon the accused persons for a defence, immediately acquitted them of the charge.

This case shows the great danger to society of receiving evidence on important pathological questions from men who perhaps take only a chemical view of such matters. A darkened patch of blood in the stomach was pronounced to be gangrene, while the yellow color of the arsenic which surrounded it, proved that putrefaction must have occurred; and red blood effused at the time of death would have been darkened by the same process which rendered the arsenic yellow. Gangrene is a rare appearance in poisoning by arsenic, and is always preceded by violent inflammation of some days' standing. Here there was an absence of inflammation of the stomach (where the arsenic was found) and of the upper part of the bowels. No calomel was detected in the stomach, and yet it was assumed by the chemist that a large dose of calomel had been given. Had the arsenic been in the body as alleged, for two days, it would have been found in the intestines, the liver, and tissues. Assuming that it was not contained in these parts, the allegation that it had been in the body for so long a period as the chemist stated, would have been proved to be utterly untrue, and the innocence of the accused made at once manifest; yet for some unexplained reason, this most necessary part of the analysis was not made! The lives of these females were thus placed in jeopardy by a series of blunders and omissions of the grossest kind. The keeping of white arsenic in powder in a shop near to calomel, is fortunately not a common practice. The employment of persons to dispense medicines, who admit that they have not sufficient knowledge of drugs to distinguish calomel from arsenic, is in accordance with the present state of the law. The same may be said of the employment of those who, with only a limited experience in chemical analysis, undertake to answer any pathological question, however difficult, which may arise in the course of an investigation.

The longer life is protracted after the supposed administration of poison, the more difficult becomes the decision in reference to the cause of death. It will be seen from the facts above related, that a question of this kind can only be satisfactorily settled, when death is recent, by a reference to the particulars attending each case. The following, which occurred to Dr. Christison, is in this respect interesting. A man named Munn was tried at the Inverary Spring Circuit, for the double crime of procuring abortion, and of murder by poisoning. The moral evidence and symptoms together, left no doubt that arsenic had been given; and that the deceased, a girl with whom the prisoner had cohabited, had labored under the effects of this poison in a very aggravated and complex form for twelve days. After that, she began to recover rapidly, and in the course of a fortnight was free from every sympton, except weakness and pains in the hands and feet: in short, all things considered, she was thought to be out of danger. But she then became affected with headache and sleeplessness, and died in nineteen days more under symptoms of obscure general fever, without any local inflammation. The medical opinion given was, that granting the girl's first illness, as it appeared from moral and medical evidence, to be owing to arsenic, her death could not be ascribed to that poison with any certainty. (On Poisons, p. 56.)

This question may sometimes present itself to a medical witness under another form, namely, whether a person has died from a medicine exhibited in an improper dose, or from disease. Thus a person enfeebled by age or disease, may be killed by a powerful drastic purgative. Infants may be killed by small doses of opium or calomel. Several lives have been already lost by the effects of repeated doses of gamboge and aloes, exhibited in large quantities to enfeebled persons, under the form of Morison's pills; and convictions for manslaughter have taken place on this ground. The questions here will be, 1. Whether the medicine or the disease caused death; or, 2. Whether the medicine accelerated death, by aggravating the disease or rendering it fatal. The responsibility of the accused will depend upon the answers: and it will be for a jury to consider whether there were sufficient knowledge and caution employed by the person prescribing it. The most simple remedies, used at an improper time, or in improper doses, may act as poisons, and destroy life. Such cases are commonly too well marked to admit of much difficulty in deciding on the real cause of death.

3. Death may have been caused by violence and not by poison. —A person who has taken poison may be maltreated, and the question will arise, whether the poison or the maltreatment was the cause of death. The solution of this question cannot in general be very difficult, when the history of the case is before us. Two instances of this kind are on record. Wildberg was called upon to examine the body of a girl, who died while her father was chastising her for stealing. It was supposed by all, that the girl had died from the effects of the violence. On the arms, shoulders, and back, many marks of violent treatment were found, and under some of them blood was extravasated in large quantity. The injuries, although severe, did not appear sufficient to account for the sudden death; he therefore proceeded to examine the cavities, and on opening the stomach, he found it very much inflamed, and lined with a white powder, which was proved to be arsenic. It turned out that on the theft being detected, the girl had taken arsenic for fear of her father's anger: she vomited during the flogging, and died in slight convulsions. Upon this, Wildberg imputed death to the arsenic, and the man was exculpated. A woman at Berlin, who had lived on bad terms with her husband, went to bed in perfect health, but soon afterwards her mother found her breathing very hard, and, on inquiring into the cause, discovered a wound on the left side of the breast. A surgeon was sent for, and the hemorrhage, which was slight, was arrested; but the woman died towards morning. On inspection, it was found that the wound had penetrated the pericardium, without touching the heart, and that the bleeding had been caused by a division of one of the intercostal arteries; but very little blood was effused in the chest. Coupling these circumstances with the trifling loss of blood during life, and the fact that she

had had much vomiting and some convulsions immediately before death, it appeared to the medical examiner that she could not have died from the wound. On a further inspection of the body, signs of corrosion and irritation were found in the mouth, throat, and stomach, and the remains of some nitric acid were discovered in a glass in the room. The facts proved that she had died from poison. (On Poisons, p. 48.) Dr. Geoghegan has communicated to me a case which occurred in Ireland in 1853, in which a gentleman having taken eleven grains of strychnia, threw himself out of a window, and sustained great bodily injury. There was so much more spasm than could be accounted for by the nature of the injury, that this led to inquiry, and it was ascertained that he had purchased and taken strychnia, and that this was the cause of death. A man was found hanging in a room which was locked on the inside. On an inspection of the body, a large quantity of prussic acid was found in the stomach. This was an act of suicide. The deceased had swallowed the poison after having adjusted the rope round his neck, and had had sufficient power to throw himself from the stool on which he was standing. The cause of death may be easily assigned in such cases when the circumstances are known; but it is evident that without great care in conducting examinations, the apparent may be mistaken for the real cause.

The kind of violence may sometimes sufficiently account for death without reference to the poison which may have been taken. The following case occurred in 1836. A young man was found hanging in his bed-room, quite dead. He was suspended by his cravat, and his feet were within an inch of the floor. The door of the room was fastened on the inside, and it was proved that no one could have had access to it. An earthen pan was found near the bed, containing about a pint of blood, which appeared to have proceeded from a very deep incision in the bend of the left arm of the deceased. The razor with which this had been inflicted was found on the mantelpiece. It came out in evidence, that on the night previously, the deceased had swallowed a quantity of arsenic, and had suffered severely from the effects of the poison; although, at the time, it was supposed that his illness was due to other causes. In this case there were three modes by which suicide was attempted. The deceased had first taken poison, then wounded, and afterwards hanged himself. There could be no doubt that death was caused by hanging; and had the wound been inflicted, and the poison administered by other parties, this opinion might have been safely expressed. Had the body been found hanging in a suspicious locality, these circumstances might have created a strong presumption of murder. Casper met with a case in which an aged female was found dead, with a soft cloth round her neck, under circumstances which gave rise to a suspicion that she had died from strangulation. The cloth, on examination, was found to have on it spots corroded by sulphuric acid. There was no mark on the neck; but there were brown streaks of a leathery consistency, as from a corrosive liquid, at the corner of the mouth and on the neck; the lungs were healthy and the heart empty. The lining membrane of the gullet was of a gray color, and that of the stomach presented a similar appearance. The coats were soft. They had an acid reaction, and free sulphuric acid was found both in the stomach and gullet. The blood was feebly acid. These facts left it certain that death had taken place from oil of vitriol, and not from strangulation. (Handb. der Ger. Med. p. 427.)

A singular case is reported by M. Desgranges, in which a man was found lying on the ground in an insensible state, with marks of contusions on his body as if he had fallen from the window of his room. He died without recovering his senses. On inspection, a large quantity of carbonate of copper was found diffused through the whole of the alimentary canal. From the facts which came out, the cause of death was referred to this poison. (Med. Gaz. xxxi, 495.)

The real cause of death may not, however, be always so clear; for a severe wound, sufficient to account for death, may have been inflicted on the person who has taken poison. A case occurred to Mr. Watson, of Edinburgh, which may serve as an illustration. A woman, aged 60, was brought into the Royal Infirmary, on the evening of the 7th February, 1838, having her throat extensively cut: she died shortly after admission. It was ascertained that she had swallowed two ounces of sulphuric acid, a quarter of an hour before cutting her throat. After having taken the acid, she was seen writhing in great pain; she had then put a razor into her pocket, and left the house to cut her throat. She inflicted the incisions on arriving in the street, was immediately seen, and conveyed to the Infirmary, which was close by. She died in about half an hour after taking the sulphuric acid. (Ed. M. and S. J. April, 1840.) The wound in the throat was very deep, and besides other vessels, it divided completely the internal jugular vein on the left side. At the inspection, there being then no suspicion of poison, it was supposed that the bleeding from the wound sufficiently accounted for death. On opening the abdomen, three-fourths of the stomach were wanting, its coats having been dissolved and decomposed by the action of the sulphuric acid. Whether this or the bleeding was the cause of death, it was rather difficult to say: but probably the loss of blood, by weakening the system, accelerated the effect of the shock produced from the extensive injury to the stomach by a corrosive poison. Thus both causes may have operated, since it is unusual for sulphuric acid to destroy life within so short a period of time. I need hardly observe, that had the wound been inflicted by another, an important question would have arisen respecting the degree of criminality to be attached to the party who had inflicted it. (For some practical suggestions on mixed cases of this kind, see Belloc. Cours de Méd. Leg. 148.)

4. OF TWO POISONOUS SUBSTANCES TAKEN BY THE DECEASED, WHICH CAUSED DEATH.—This question does not relate so much to the subject of compound poisoning, as to cases of the following kind, which may require careful medical investigation. A person may have poison administered to him while laboring under the effects of powerful medicine, or of some other poison. Thus a patient, while under a course of mercury, may have had corrosive sublimate administered to him with intent to murder. After a certain period, violent salivation with sloughing may ensue, and the patient die. Is death in such a case to be ascribed to the corrosive sublimate, or to the mercurial medicine previously adminis-

tered? It may be necessary to state, that death is sometimes a result of the severe salivation induced by preparations of this metal, prescribed medicinally, in a mild form and in small doses.

In Butterfield's case, tried at Croydon many years ago, this question incidentally arose. The prisoner was indicted for administering corrosive sublimate to the deceased. The immediate cause of death was profuse salivation: and this was referred, by the medical witnesses, to the operation of the poison. It was proved, however, in the defence, that about two months previously to this attack, the deceased had been under treatment with some quack medicine, by which he was violently salivated; but this salivation had entirely ceased, and during the whole of the above-mentioned period he had abstained from taking any mercurial preparation. It was at this time, that the corrosive sublimate was supposed to have been secretly administered to him in small doses. The prisoner, however, was acquitted of the charge, on the ground that, as mercury had been introduced into the system of the deceased by a quack medicine, the fatal salivation might have proceeded from a recurrent operation of this medicine, and not from the poison. The case of Lacoste raised a similar question with respect to the medicinal and criminal use of arsenic (ante, p. 50).

A woman was found dead, and near her body was a glass containing some sulphuric acid. This gave rise to a suspicion of poisoning. On inspection, the mouth and throat were covered with a black mucous matter, and the gullet was filled with a tarry-looking mass, which had a strong acid reaction. The coats were softened, and the lining membrane was easily detached. The stomach was throughout inflamed, and of a brownish-black color; its coats were so softened that it could not be moved without lacerating it. It contained a large quantity of a black viscid liquid, in which, as well as in the gullet, arsenic and sulphuric acid were detected. (Von Raimann, Med. Jahrb. 20 B. 2 S. 221.) Admitting that two poisons were taken in this case, which was taken first? Most probably the arsenic. It is more difficult to say which caused death, because the deceased was not seen during life; and probably she was already laboring under the effects of arsenic, when she swallowed the sulphuric acid. The fact of this last poison having been taken appears to show, that it was a case of suicide. But perhaps the sulphuric acid itself contained arsenic as an impurity, since some specimens prepared from arsenical pyrites, are found to be impregnated with a large quantity of this substance.

It is obvious that, for the proper investigation of cases of this description, a medical witness should be prepared with a full knowledge of the peculiar properties of most poisons,—the doses in which they prove fatal,—the power which they have of modifying each other's effects,—and the period of time within which they produce their symptoms, and commonly destroy life.

In April, 1858, a question arose at an inquest, in this metropolis, whether certain persons found dead in a house which was burnt, had died from the effects of suffocation from smoke, or from the fumes of white arsenic, evolved, as it was alleged, under peculiar circumstances. Fourteen bodies, found dead in the house, underwent a medical inspection; in five of these analyses were made, and arsenic was discovered, as it is stated, in the blood, lungs, stomach, and some of the air-passages of four. The quantity of arsenic thus said to have been found was not specified; it appears in the report of the proceedings merely as "distinct traces." (Med. Times and Gazette, April 24, 1858, p. 429.) In four of the bodies, the blood was of a light or bright-red color. In some (case of William Hedger), the blood was dark-colored, and, in this case, death was ascribed to suffocation, although arsenic is stated to have been found in the blood! In the four cases above-mentioned death was referred by the chemical witness to the poison of arsenic, inhaled in fumes, as a result of the fire having extended to certain arsenical minerals in a museum adjoining the burning premises. The reasons assigned for this opinion are, however, inadmissible: 1. It is not the property of arsenic in fumes to redden the blood in the manner suggested,—the exceptional case referred to, proves that arsenic may exist in the blood without producing such an effect. 2. It is not the property of arsenical fumes to destroy life with the suddenness with which these persons must have died. 3. More than "distinct traces" of arsenic would have been required to produce any effect, and the rapid diffusion of the vapors and the fumes in the heated currents of air would have rendered them harmless compared with the effects instantaneously produced by the gaseous products of combustion. 4. The statement that such traces of arsenic, as are said to have been breathed, would act as a narcotic, and produce immediate prostration of strength, is unsupported by any known facts.

The evidence given by the non-chemical witnesses at this inquiry leaves it clear that the deceased perished from the usual cause in burning houses,—the inhalation of the products of combustion. One of these, carbonic oxide gas, has been proved, by Bernard, to produce the light-red color of the blood which puzzled these "experts," and bewildered the jury. No mineral poison diffused in vapor in the air can act so quickly as carbonic acid or carbonic oxide in extinguishing life; and it is obvious, that these destructive gases must have been produced, and caused the fatal effects before the minerals could have been heated to a sufficient temperature to diffuse any injurious quantity of arsenical vapor. The jury, however, appear to have been easily satisfied by this extraordinary evidence. They referred the deaths of the four persons with light-red blood to the mortal effects of certain poisonous fumes and common wood-smoke, and of the one person (with black blood) to suffocation by smoke, although the blood of this man was said to contain arsenic!

5. DEATH MAY BE CAUSED BY POISON AND NO POISON BE FOUND IN THE BODY.—The circumstances under which this condition may present itself have already been considered (ante, p. 175). The poison may be one which does not admit of detection by analysis. The person may have lived so long that it has been entirely eliminated, or the body may have been buried for so long a period that all traces of it are lost. In these cases evidence of death may be supplied by symptoms and appearances, and this evidence may be supported by moral circumstances, showing that the accused had the means, the motive, and the opportunity of administering the poison.

CHAPTER XIV.

CONCLUDING REMARKS ON GENERAL POISONING—MORAL AND CIRCUMSTANTIAL PROOFS—STATISTICS OF POISONING—CORONERS' RETURN OF DEATH FROM POISON—OTHER RETURNS—RETURNS FROM HOSPITAL PRACTICE—INCREASE IN THE NUMBER OF DEATHS FROM POISON—DEATHS FROM POISON IN FRANCE AND DENMARK.

THE duty of a medical witness, as such, is accomplished when he has proved, on a charge of criminal poisoning, that death was *certainly* due to poison. The moral and circumstantial evidence must prove that the accused was the party who gave it;—this proof often fails,—the fact of administration cannot be brought home to the accused, and the case falls to the ground. It is not within the province of this work to treat of moral and circumstantial evidence in cases of poisoning. Proofs of this kind, it is true, are sometimes very closely mixed up with the evidence of professional witnesses, and in the foregoing chapters some of these have been already adverted to. A witness must, however, be cautious not to base his opinion, in questions of poisoning, on moral and circumstantial proofs. He is called upon to give a medical opinion of the cause of death, and from *medical* facts only. The moral and circumstantial proofs refer chiefly to the *administration* of poison by a particular party, and the *intent* of the person charged with the crime: it is therefore considered to fall within the province of a jury alone to decide on their relevancy and value, although it must be confessed, that many of these facts can only properly be estimated by persons versed in medical science. Supposing death by poison to have been clearly proved, it may be necessary to ascertain whether the act was the result of *accident*, *suicide*, or *homicide*. This is a question also for a jury to determine, and not for a witness; although its solution often depends upon a proper appreciation of medical circumstances. Suicide or murder may sometimes be inferred, according to the medical evidence given of the effects of certain poisons. Some speedily annihilate volition and the power of locomotion, and therefore render it a question of serious difficulty, whether particular acts could or could not have been performed after the deceased had taken the poison. On the answer to this question may depend the acquittal or conviction of a person charged with the crime.

There is one peculiarity in the legal consequences of the act of killing by poison, namely, that the act itself is generally considered in law to furnish evidence of malice. If a poison is knowingly administered to another, with the intention of destroying life, the crime is never reduced to manslaughter,—whatever may have been the provocation which the party administering has received from the person whose life he has thus taken. It is not necessary, therefore, that any particular enmity should be proved to have existed between the prisoner and deceased, although this often weighs as a strong moral circumstance against the former;

and the absence of any apparent motive for the crime, on the other hand, is always regarded as a strong presumption in favor of the accused. When a man is killed by a wound in a quarrel, the law will sometimes find an excuse for the act, from the heat and passionate excitement under which the aggressor was laboring at the time; but if the aggressor should avenge himself by secretly administering poison to his adversary, there is no excuse for the act, since it evinces cool, reflecting, and deep-rooted malice. That death by poison should ever amount to manslaughter, therefore, it must be shown, that the substance was administered to or laid in the way of the deceased by mistake, or with innocent intention; and the proof of this always lies with the accused—the law inferring that malice exists until the contrary appears from the evidence. Whether malice exists or not, is, however, in general soon made apparent from the evidence for the prosecution. (See ante, p. 26.)

In relation to medico-legal practice, the *statistics* of poisoning are of some interest. They indicate to a medical jurist, the substances which are most frequently selected for the purposes of suicide and murder, and with the properties of which, it will be expected that he should be acquainted. Unfortunately, very few tables of this kind have been published; and those which have appeared are defective in many points. One of the most complete is that which was published by order of the House of Commons from returns made by the coroners of England, of the number of inquisitions held during the years 1837 and 1838, wherein death was caused by poison. The following is an abstract of the paper, which appeared in the Medical Gazette for November, 1839.

The number of deaths by poison (returned) in the two years above mentioned, were, exclusive of two cases of suffocation by gases,—541, of which number 282 were males, and 252 females. The substances which caused death, may be taken in the following numerical order.

Opium	Laudanum,	133			
	Opium,	42			
	Other preparations,	21			
			196		
Arsenic,			185		
Sulphuric acid,			32		
Prussic acid,			27		
Oxalic acid,			19		
Corrosive sublimate and mercury,			15		
Mixed or compound poisoning,			14		
Oil of bitter almonds,			4		
Poisonous mushrooms,			4		
Colchicum, nux vomica (of each, 3),			6		
Nitric acid, caustic alkali, tartar emetic, acet. morphia, strychnia, deadly night-shade, aconite (of each, 2),			14		
Bichrom. potash, nit. silver, Goulard's extract, sulph. iron, mur. tin, hellebore, castor-oil seeds, savin, hemlock, cantharides, cayenne pepper (of each, 1),			11		
				527	
Unknown,				14	
					541

[A little discrepancy exists in the relative number of cases, probably owing to the fact that in several instances some of the poisons were taken in a compound state. The reader will find the details in the Med. Gaz. vol. xxv, p. 204.]

It will be seen by this table, that the largest proportion of cases of poisoning in England are those by *opium* and *arsenic:* the greater number of the former, being cases of suicide and accident, and of the latter, cases of criminal poisoning. There can be no doubt that the number of deaths from poison which annually occur in England and Wales, are much greater than this table represents. The annual registration of deaths, although defective with respect to the number of individual poisons,—two only, *i. e.* arsenic and opium being commonly recorded, and these imperfectly,—shows that the mortality from this cause, including overdoses of medicine, is greater than is commonly supposed. I here subjoin a table of the deaths from poison, in 1840, drawn up from the sixth Annual Report of the Registrar-General (1844). The deaths from this cause in 1840, are stated to have been 349, of which number there were 181 males and 168 females. The cases of suicide from poison were 161, being 87 females to 74 males,—the cases of accident or homicide were 188, being 107 males to 81 females. Of the 75 cases of poisoning by opium, 42 occurred in children under five years of age,—a lamentable proof of the extensive mortality among children from the improper administration of this drug. These cases occur among the returned deaths from opium; but under the head of medicines improperly administered, three-fourths of the deaths took place among children under five years of age!

Opium, ...	75
Arsenic, ...	32
Other poisons, including medicines improperly administered, . . .	242
Total deaths from poison in 1840, ...	349

It would be a considerable benefit to medical science, if the poisons which caused death were more distinctly specified in the Registration returns. The poison or medicine should be stated as distinctly as in the returns now made with respect to fatal diseases. The cases of poisoning by arsenic and opium in the above table are evidently understated.

A proper collection of hospital statistics would throw great light on the frequency of death from poison, and the nature of the poison taken. Mr. Wilson ("The Bane and the Antidote," Birmingham, 1856) states that, in sixty-three cases of poisoning treated in the Birmingham Hospital from 1848 to 1856, a period of eight years, the nature of the poison used was as follows:

Opium or laudanum, 12; sulphuric acid, 9; oxalic acid, 8; acetate of lead, 5; nitric acid, 4; arsenic, 3; sulphate copper, 2; iodine, 2; nux vomica, 2; ammonia, 2; cyanide potassium, 1; corrosive sublimate, 1; strychnia, 1; essence of bitter almonds, 1; muriate of morphia, 1; charcoal-vapor, 1; other poisons, 8; total, 63. The number of females was 42, and of males, 21. The deaths were only 5.

This table indicates that the number of deaths from poison are few compared with the actual cases of poisoning. Probably, on an average, not more than one in five cases proves fatal.

I am indebted to Dr. Steele, Superintendent of Guy's Hospital, for some statistical information on this subject, as collected from the records of Guy's Hospital during the years 1854, 1855, 1856.

	Admitted.	Died.	Recovered.
Males,	11	4	7
Females,	16	7	9
Total,	27	11	16

Arsenic, 2; sulphuric acid, 3; nitric acid, 1; oxalic acid, 4; laudanum, 5; prussic acid, 1; oil of bitter almonds, 1; solution of ammonia, 1; brandy, 1; chloride of zinc, 1; poisonous berries, 2; locust beans, 1; oxide of mercury, 2; unknown, 2; total, 27.

Of the sixty-three cases reported from the Birmingham Hospital, five proved fatal. This is in the proportion of about 8 per cent., or one death for twelve cases. Among the twenty-seven cases at Guy's Hospital, there were eleven fatal cases, making a proportion of 40 per cent. or two deaths for every five cases. The average annual admissions at the Birmingham Hospital were eight; at Guy's Hospital, nine cases.

Mr. Wilson has also published an analysis of the *deaths* from poison in England alone, taken from the Registrar-General's Report for six years, from 1848 to 1853,—the latest returns then published. The total deaths, as recorded in these returns during the period above mentioned, were 3218; distributed as follows:

	Males.	Females.	Total.
1848,	308	261	569
1849,	290	236	526
1850,	304	249	553
1851,	275	253	528
1852,	253	300	553
1853,	270	219	489
Total deaths in six years,	1700	1518	3218

This gives an average annual mortality from poisons for England alone, of 536; but the frequent exhumations of bodies, buried under erroneous certificates, prove that the mortality must be greater than these figures indicate. Some cases escape the coroner's inquisition entirely. From the table of returns, elsewhere published (ante, p. 219), it will be perceived that, while the deaths from poison in two years (1837–8) for England and Wales, amounted to 541, in 1848, *i. e.* ten years later, the number for this year only, amounted to 569. Assuming the returns of the Registrar-General to be correct, the increase had therefore been more than 100 per cent., and the deaths in 1840, compared with those in 1850, had increased from 349 to 553. But the cases of poisoning cannot be estimated by the number of deaths. Assuming the average mortality to be not more than one out of three cases, they would give a total of 1600 cases of poisoning per annum. The relative proportions in which the deaths are occasioned by the various poisons, can be computed at present only from the coroners' returns of 1837–8. The poison was ascertained in 527 cases. Of these opium and its preparations formed 37 per cent.; arsenic, 35 per cent.; sulphuric acid, 6 per cent.; prussic acid, 5 per cent.; oxalic acid, 3·5 per cent.; and mercurial preparations about 8 per cent. Opium and arsenic were the causes of death in three-fourths of the whole number of cases, and opium caused a greater number of deaths than arsenic.

The subjoined tables represent the statistics of poisoning in Denmark from the year 1830 to 1835 (Med. Gaz. xxv, 575); and following this is a table of 94 cases observed in France, during a period of seven years, 1825–32 (Briand, p. 434).

IN DENMARK.

Sulphuric or nitric acid, generally diluted,	74
Arsenic,	16
Caustic alkalies,	5
Opium,	2
Litharge, verdigris (of each 1),	2
	99

IN FRANCE.

Arsenic,	60
Fly-powder,	3
Verdigris,	7
Corrosive sublimate,	5
Cantharides,	5
Nux vomica,	4
Nitric acid,	2
Acet. lead, carb. lead, sulph. zinc, tartar emetic, opium, prussic acid, mercurial ointment, orpiment (of each 1),	8
	94

It is difficult to compare these two tables with the preceding,—the number of observations being much fewer. Poisoning by arsenic is, however, proved by them to be very common. Out of 616 charges of poisoning in France, during a period of twenty years, one-half, if not two-thirds, were cases of poisoning by arsenic. (Flandin, Des Poisons, i, 448.) It is remarkable that the mineral acids should have caused so many deaths in the kingdom of Denmark, the proportion being no less than three-fourths of the whole number.

IRRITANT POISONS.

NON-METALLIC IRRITANTS.

CHAPTER XV.

DIVISION OF IRRITANT POISONS—OIL OF VITRIOL OR SULPHURIC ACID—SYMPTOMS CAUSED BY THIS POISON—SOMETIMES PRODUCES SUFFOCATION—ACTION OF THE DILUTED ACID—TIME AT WHICH THE SYMPTOMS BEGIN—CAN THEY CEASE AND REAPPEAR?—APPEARANCES OF THE BODY IN ACUTE AND CHRONIC CASES—QUANTITY OF ACID REQUIRED TO DESTROY LIFE—FATAL DOSES—PERIOD AT WHICH DEATH TAKES PLACE—TREATMENT—CHEMICAL ANALYSIS—MODE OF DETECTING THE POISON IN PURE AND MIXED LIQUIDS—THE ACID NOT ALWAYS FOUND IN THE STOMACH—ITS DETECTION IN ARTICLES OF CLOTHING—AROMATIC SULPHURIC ACID—SULPHATE OF INDIGO—QUANTITATIVE ANALYSIS.

General remarks.—IRRITANT POISONS may be divided into four groups,—the non-metallic—the metalloids—the metallic—and those of an organic nature, *i. e.* derived from the vegetable and animal kingdoms. The non-metallic irritants comprise the mineral acids, oxalic acid, the alkalies, and their salts. According to strict chemical views, the alkalies and their salts should be placed among the metallic irritants; but it will be, in many respects, convenient to consider them in the same group with the acids. Besides, although they certainly have metallic bases, the demonstration of the existence of the metal is never required at the hands of a medical jurist, as in the case of the true metallic irritants. Among the mineral acids, we shall first speak of poisoning by sulphuric acid.

OIL OF VITRIOL, SULPHURIC ACID.

This is met with in commerce in two states, either concentrated or diluted. The concentrated acid is a heavy oily-looking liquid, often of a brown color; it has a strong sharp acid taste—it powerfully reddens vegetable colors, and corrodes and destroys most kinds of organic matter.

The term oil of vitriol is strictly applied only to sulphuric acid, which has an oily consistency and a great specific gravity (from 1·800 to 1·845). It was so called because it was obtained by the distillation of green vitriol, of which it was considered to be the oil or spirit. It is in this state eminently corrosive, and this corrosive property is lost when the oily consistency is removed by dilution. Oil of vitriol is under all circumstances sulphuric acid, but sulphuric acid is not in all cases oil of vitriol. The question has been raised whether an acid of a sp. gr. of 1·420 should be regarded as oil of vitriol. Such an acid contains 56 per cent. of water; it has no oily consistency, and has none of the well-marked chemical (corrosive) properties of oil of vitriol.

Sulphuric acid is very frequently taken as a poison by suicides: but probably there is no case in which the sufferings of the individual before death are more intense. In medico-legal practice, it is not common to find that this acid is employed for the purpose of murder. Children have, however, been destroyed by a quantity of it being poured down the throat; and it is obvious that a person who is drunk or asleep, may be thus easily killed. (Case of Humphreys, ante, p. 113.) With these exceptions, which are of rare occurrence, instances of fatal poisoning by sulphuric acid, may be pretty equally divided into cases of suicide and accident. The taking of this liquid is a frequent form of self-destruction among females;—less frequent among males, and by no means uncommon as an accident among young children of both sexes. On the discovery of a dead body, poisoned by sulphuric acid, a medical jurist will have, then, especially to consider the age of the deceased. If it be a new-born child, or an infant, it is certain that the poison has been homicidally or accidentally administered; if a child, all other circumstances being equal, that it has been swallowed by accident; if an adult, that it has been voluntarily taken for the purpose of suicide. It is to be observed, that there is no poison which can be obtained more readily or without exciting less suspicion than sulphuric acid, since it is used for many domestic purposes. The only probable case of murder by this poison in an adult, would be where the person was either intoxicated or asleep when it was administered; but even then the individual would be immediately roused. It is not easy to imagine that a criminal, who wished to destroy the life of another, would attempt this by causing him to swallow forcibly a quantity of oil of vitriol, when there are so many other more ready, secret, and speedy means of destruction at hand. It is also impossible that such a substance as this should, like arsenic, be secretly administered in articles of food. Its powerfully acid taste in the smallest quantity, and the fact that the physical qualities of the food would in general be changed by the chemical action of the acid, would certainly lead to a discovery and frustrate the attempt. There are but few instances in which such an attempt to poison, has been made. In one of these, a boy being offended with his mistress, put a quantity of common diluted vitriol into a cup of tea, which she was about to drink: in another, the attempt to administer was made by putting the acid into coffee. In both instances the taste immediately led to the discovery of the attempt.

SYMPTOMS.—*The Concentrated Acid.*—When this poison, which is one of the most powerful corrosives, is swallowed in a concentrated form, the symptoms produced come on *immediately*, or during the act of swallowing. There is violent burning pain extending through the throat and gullet to the stomach—the pain is often so severe, that the body is bent. There is an escape of gaseous and frothy matter, followed by retching and vomiting, the latter accompanied by the discharge of shreds of tough mucus and of a liquid of a dark coffee-ground color, mixed or streaked with blood. The mouth is excoriated, the lining membrane and surface of the tongue white or resembling soaked parchment,—in one instance the appearance of the mouth was as if it had been smeared with white paint; after a time, the membrane acquires a gray or brownish color; the cavity is filled with a thick viscid phlegm, rendering speaking and swallowing very difficult. If the poison has been administered by a spoon, or the phial containing it has been passed to the back of the throat, the mouth may escape the chemical action of the acid. A medical witness must bear this circumstance in mind, when he is called to examine an infant suspected to have been poisoned by sulphuric acid. Around the lips, and on the neck, may be found spots of a brown color from the action of the acid on the skin. There is great difficulty of breathing, owing to the swelling and excoriation of the tongue and throat; and the least motion of the abdominal muscles is attended with increase of pain. The abdomen is distended and tender. These symptoms, although peculiar and well marked, have been sometimes mistaken for those of disease. (Henke, Zeitschrift der S. A. 1843, ii, 284.) The stomach is so irritable, that whatever is swallowed is immediately ejected, and the vomiting is often violent and incessant. In a case which occurred to Dr. Geoghegan, the patient (a female) vomited for three or four hours. This symptom then ceased, and did not reappear, although she lived thirty-one hours (Med. Gaz. vol. 48, p. 328). Vomiting, although a common symptom, is not always immediate. A case is reported in which a man, æt. 30, swallowed two ounces of oil of vitriol (1·842), and died in twenty-five hours. Half an hour after he had taken the poison he resembled a patient in the collapsed stage of cholera. The inside of the lips, tongue, and throat were swollen, and had the appearance of being smeared with thin arrow-root. He suffered severe pain, but did not vomit until *three-quarters of an hour* had elapsed; the vomiting appeared to be then excited by the liquid given to him. The vomited matters were dark, bloody, and viscid. (Ed. Monthly Jour. 1850, p. 538.)

The matters *first* vomited generally contain the poison: they are acid, and if they fall on a limestone pavement there is effervescence; if on colored articles of dress, the color is sometimes altered to a red; or (if logwood) yellow, the color is discharged and the texture of the stuff destroyed: on a black cloth dress, the spots produced by the concentrated acid are brown, with a fringe of red, and they remain moist for a considerable time. An attention to these circumstances may often lead to a suspicion of the real cause of the symptoms, when the facts are concealed. In a case of attempted murder by sulphuric acid in beer, the nature of the poison was suspected from the beer having corroded an apron on which a portion had become accidentally spilled. After a time, there is great exhaustion, accompanied by general weakness: the pulse quick and small; the skin cold, and covered with a clammy sweat. There is generally great thirst, with obstinate constipation of the bowels; should any evacuations take place, they are commonly either of a dark-brown or leaden color, in some instances almost black (carbonaceous), arising from the admixture of altered blood. They also contain corroded portions of the lining membrane of the bowels. There are sometimes convulsive motions of the muscles, especially of those of the face and lips. The countenance, if not livid from obstructed respiration, is pale, expressive of great anxiety, and of dreadful suffering. The intellectual faculties are quite clear, and death usually takes place very suddenly, in from eighteen to twenty-four hours after the poison has been taken.

The Diluted Acid.—When the acid is diluted, the symptoms are of the same character but less severe, and not so quickly produced. They vary according to the degree of dilution, the poison acting only as an irritant when much diluted. The vomited matters are not so dark-colored: in one instance they were nearly colorless. It may be proper here to state, that the diluted sulphuric acid of the London Pharmacopœia has a sp. gr. of 1·103. A fluid-ounce contains sixty grains of anhydrous sulphuric acid, or by weight 12·43 per cent. The corrosive properties of the acid are destroyed by dilution, but its irritant properties are retained.

Within what period of time do the symptoms commence?—Most toxicologists, including Orfila (Toxicologie, i, 83, 1843), Christison (On Poisons, 4th ed. 90), and Galtier (Traité de Toxicologie, i, 121, 1845), state that the symptoms commence *immediately*, or during the very act of swallowing, *i. e.* a sense of heat is experienced, with excoriation and burning pain in the throat and stomach. Considering the powerful chemical action of the poison on the thin mucous membrane of the mouth and fauces, it is not easy to understand how there should be any delay in the production of some visible symptoms. In rabbits I have always observed instantaneous effects on the contact of the acid, such as foaming and frothing at the mouth, with a milky-white appearance from the action of the poison on the lining membrane. In most cases that have hitherto been accurately noticed from the commencement, *i. e.* from the act of swallowing, there has been immediately an escape of gas, with severe retching, followed within a short period by vomiting. The question relating to the time of occurrence of symptoms was of some importance at the trial of the Queen v. North (Guildford Summer Ass. 1846); for upon the answer to it, rested, in some measure, a charge of murder. (Guy's Hospital Reports, Oct. 1846, iv, p. 396.)

From a case, observed by Orfila, it appears that, even when moderately diluted, there is no delay in the appearance of the symptoms produced by this poison. A man swallowed a certain quantity of sulphuric acid, diluted with its weight (*i. e.* with twice its bulk) of water, and experienced immediately the most severe suffering. (Toxicologie, i, 96.) The common opinion of toxicologists, that this poison, from its local chemical action, produces at once certain effects, is, I believe, correct in all cases in which it is not much diluted with water. It causes some immediate symptoms, and in most cases early vomiting,—a fact borne out by the results of the repeated experiments on animals. A case is reported in the Medical Gazette (vol. xxxix, p. 147), from which it appears that half a teaspoonful of sulphuric acid was given by mistake for castor oil to a child a year old, that certain symptoms immediately followed: the child cried and was restless, and this led to the discovery of the mistake.

In a case, tried at the York Assizes in July, 1846 (Reg. v. Rodda), a man was convicted of the murder of his infant child by the administration of oil of vitriol. The moral circumstances showed that the poison had been procured by the prisoner; that the infant was not likely to have taken it itself; and, although the child had been frequently fed during the evening, the commencement of the action of the poison was traced to a few minutes during which the child was left alone in the care of the prisoner. Here the medical evidence had a very important bearing, for it was properly insisted that the visible effects caused by oil of vitriol are almost instantaneous, and admit of no delay in their production. The prisoner's defence was, that he was quite innocent of the charge, and that he had neither bottle, nor spoon, nor anything else in the house; that he put the child into the crib, and that it began to vomit; its mother came down stairs,—the child was then throwing up something, and he went for a doctor. The prisoner was convicted, and executed. The reader will here perceive, that the administration was clearly brought home to the prisoner by this well-known effect of the mineral acids in producing immediate symptoms.

The local action of sulphuric acid on the mouth, throat, and gullet, is generally very energetic: the lining membrane is stripped off in shreds, or peels off in large masses. In a case, mentioned by Sobernheim, the lining membrane of the mouth, tongue, and throat, came off in one mass. In another, related by Dr. Wilson, the patient, during a violent fit of coughing, brought up a large piece of sloughy membrane, which was found to consist of the inner coat of the gullet much thickened and very firm in texture. Its length was eight or nine inches, and its width that of the gullet; it was of a cylindrical form, and pervious throughout its whole extent. (Med. Gaz. vol. xiv, p. 489; also, vol. xxii, p. 76.) This has been observed to occur in other cases. (See Galtier, Toxicologie, i, 199.)

The acid may not go farther than the entrance of the throat. In May, 1857, a man was admitted in Guy's Hospital, who, about four years previously, had, by mistake for beer, taken into his mouth a tablespoonful of oil of vitriol. On finding that it had an acid and burning taste, he ejected it, and none was swallowed. He immediately took some milk, with chalk and water. There was no vomiting, until an emetic was given. He complained of a burning pain in the mouth and throat: he articulated indistinctly, and speaking was painful to him. The lining membrane was covered with a thick whitish fur, which was peeling off in flakes, leaving a reddish-brown surface. The teeth presented a blackish-yellow appearance. His mouth was filled with a frothy, viscid, tenacious fluid, containing shreds of mucous membrane. It was not acid. The attempt to swallow produced severe pain and convulsive cough, and the fluid sometimes returned through the mouth and nostrils. When food was taken, there was a feeling of sickness in the stomach, but no vomiting. The man progressed favorably. In fourteen days he left the hospital with his mouth and throat restored to their natural condition.

The acid produces asphyxia.—This poison may destroy life without reaching the stomach,—a fact sometimes observed in children. The larynx is then acted on: the air-passage is closed by the swelling of the surrounding parts, and the child dies suffocated. In such cases death takes place very rapidly. I have found that rabbits, to which this poison was given, died from this cause in the course of a few minutes. Mr. Quain met with the case of a child which became asphyxiated under these circumstances, while he was performing the operation of tracheotomy. The child revived on inflating the lungs, but died three days afterwards of bronchitis. On inspection, it was found that the acid had not even reached the gullet. (Lancet, Oct. 29, 1836.) Owing to this local action on the air-passages, sulphuric acid may easily cause death by suffocation. (See case by Dr. A. T. Thomson, Lancet, June 10, 1837.)

On the other hand, Ryland and Porter have remarked that in *suicide* by sulphuric and other mineral acids, the larynx generally escapes injury. In their view, the epiglottis, during the act of swallowing, completely covers the upper part of the glottis, and thus the acid passes down the gullet without affecting the larynx. When the acid has been swallowed by mistake, or when forcibly administered, the larynx is liable to be affected; for so soon as the mistake is discovered, which is almost immediately, all the muscles of the fauces and throat are spasmodically affected, and the fluid is ejected, partly by the mouth and partly by the nostrils; while, perhaps, a few drops pass at the same time into the glottis, causing inflammation and rendering tracheotomy necessary. When the poison has been taken voluntarily, the mucous membrane of the mouth, gullet, and stomach, will present the usual effects of the acid —*i. e.* marks of inflammation and corrosion. When it has been taken by mistake, the parts chiefly injured are the mouth, throat, epiglottis, and sometimes the lips of the glottis; and when forcibly administered to children, there will be symptoms of inflammation of the larynx accompanied by difficulty of swallowing. The appearance of spots on the outside of the face and on the neck will frequently furnish better evidence of violent administration than any of the changes produced in the mouth and throat. Although external spots of corrosion may be found in any case of poisoning by this acid, their absence is a strong indication that the acid has been knowingly or consciously taken. A boy, suffering from effects of oil of vitriol, stated that a person had given it him for spirit. There was no mark on the face and lips, and as it was clear, from an inspection of the body, that a large quantity of this corrosive liquid had been swallowed, Casper justly concluded that he had taken poison knowingly and wilfully, and had then falsely accused another. The stomach was perforated, and a large quantity of a bloody mucous liquid was found in the abdomen. (Handb. der Ger. Med. i, 428.) In Dr. Walker's case (post, p. 232), although two ounces of acid were drunk from a tea-cup, there were no stains on the outside of the lips, angles of the mouth, cheek, neck, or hands, nor upon the clothing. (Ed. Month. Jour. 1850, p. 538.) Death may take place, on these occasions, from an affection of the larynx alone; the acid may not even have reached the gullet. (See Ed. M. and S. Journ. xlix, 583; also Med. Chir. Rev. xxviii, 399.) A remarkable instance of this kind has been reported by Dr. Gull (Med. Gaz. vol. 45, p. 1102, 1850). A lady was found dead in a chair holding in her right hand a small phial labelled sulphuric acid—poison. The body was in an easy attitude, half recumbent, and there had been no struggling or motion after the acid had been taken. The air-passages, including epiglottis, larynx, and trachea, were corroded; the acid had passed into both lungs, had charred them, and by acting on the subjacent ribs, had formed a crust of sulphate of lime upon the lungs. The large bloodvessels were full of solid charred blood resembling blacking. The lining membrane of the gullet and stomach was uninjured. None of the acid had passed into the stomach. Death had taken place from suffocation, and as the body was not inspected for six days, the acid may have produced much chemical action after death. Owing to this local action on the air-passages, cyanosis (blueness of the skin) has been occasionally observed among the symptoms. (Galtier, Toxicologie, i, 192.) Thus, then, as a medico-legal fact of some importance, it is certain that this poison may destroy life without reaching the stomach. We cannot, however, say that the discovery of the effects of the poison in the stomach would indicate suicide; because in cases of murder, the stomach has been found disorganized by it, evidently showing that it must have penetrated thus far. There are at least two instances on record in which this poison has destroyed life in consequence of its having been injected into the *rectum* by mistake for a clyster. In one case the patient suffered the most acute pain, and died in the course of a few hours. (Med. Gaz. xvii, 623; Annales d'Hyg. 1846, i, 366.)

Can a person who has swallowed sulphuric acid exert the powers of volition and locomotion?—The severe pain produced by a large dose of this poison is in many cases sufficient to deprive a person of the power of motion. The patient rolls on the ground in agony. Nevertheless, numerous well-observed facts prove that he may sometimes retain astonishing self-command. In the case of Mr. Schwabe, who died in twenty-four hours from a dose of six drachms of sulphuric acid, it was proved that the deceased, after having swallowed the acid, beckoned to a cabman, got into a cab, and told him to drive to his house as fast as he could. The deceased had at the time a handkerchief to his mouth, and the only circumstance noticed by the driver was, that he looked very pale. (Med. Gaz. xxxvi, 826.) A case is quoted by Dr. Galtier, in which a man, æt. 52, after having taken some soup, swallowed three ounces of commercial sulphuric acid. He threw himself upon his bed, and it was not until between three and four hours afterwards that the severe pain which he suffered compelled him to seek for assistance. He got up, dressed himself, and was conveyed to the hospital, where he died five hours after swallowing the poison. (Toxicologie, i, 189.) In another case, a child, æt. 9, swallowed an ounce of oil of vitriol, and although instantly seized with severe pain in the throat and stomach, he was able to run home and inform his parents. (Med. Gaz. xxxix, 116.) This retention of power cannot always be referred to the fact of the stomach containing food or liquid sufficient to dilute the poison: because, in Mr.

Schwabe's case, the acid was not taken until some time after a meal. In Dr. Walker's case (post, p. 232), the patient was able to get out of bed and sit on a night-stool, twenty hours after he had taken two ounces of oil of vitriol, and five hours before he died from its effects. These facts are important in a medico-legal view, as the following case will show. In December, 1843, a soldier was found lying on the pavement, and suffering from the effects of sulphuric acid. When questioned, he declared that he had been poisoned at a wine-merchant's shop. The man soon died, and the inspection showed that his death had been caused by sulphuric acid, taken probably in a diluted state. None of the acid was discovered in the matter last vomited, or in the stomach of the deceased: that which was *first* vomited had not been collected! Nevertheless, the cause of death was very clear. The wine merchant's shop where the deceased said he had been poisoned, was at some distance (not specified) from the spot where his body was found; and on the question being put to MM. Ollivier and Chevallier, they gave it as their opinion, that the deceased could not have exerted a power of locomotion for so great a distance, and affirmed that, in their judgment, based upon cases fatal within a similar period of time, the deceased could have walked only a very short distance after swallowing the poison. They therefore inferred that it was a case of suicide, and not of homicide. (Ann. d'Hyg. 1845, i, 179.) Considering the facts above detailed, and that the sulphuric acid was in this instance diluted, the medical opinion here given appears to have been somewhat stronger than prudence would warrant. A person who has taken sulphuric acid may undoubtedly retain a power of locomotion; but the degree to which it may be exerted, must depend on the special circumstances of the case.

In October, 1856, a man who had swallowed by mistake a dessert-spoonful of oil of vitriol, was admitted into Guy's Hospital. He was able to walk up stairs to his bed, and did not appear very ill, although dejected. The lining membrane of his mouth was of a brown color. He vomited slightly at first, and there was one fluid evacuation from the bowels of a brown color. On the two following days he appeared depressed, but there were no urgent symptoms. The case was considered slight, and there was every expectation that he would recover. He died suddenly on the third day. Mr. Porter mentions the case of a girl who, after having swallowed a quantity of concentrated sulphuric acid, sat quietly down to tea with some friends, although the quantity of acid taken was sufficient to cause her death in a few hours. Another case is related in which a man took a second dose of the same acid, because he thought the first might not be sufficient (Med. Chir. Rev. Vol. 28, p. 399).

Can the symptoms produced by this poison cease and reappear?—In general, it is observed that the symptoms continue to increase in severity until death, when the case is rapid: but there may be remissions, and, just before death, the pain and suffering have been observed to become considerably abated. In other cases, as in that above related, there may be an intermission of the symptoms, although the case may ultimately prove fatal. The following case, communicated by the late Dr. Johnson, to the Westminster Medical Society, in October, 1836, is in this respect singular. A person swallowed two ounces of concentrated sulphuric acid. After suffering from severe symptoms, the patient rallied and apparently recovered. A few days afterwards, during a severe fit of coughing, he brought up a quantity of the acid in its pure state: the acid having, it is said, been a fortnight in the stomach! It produced, in its passage upwards, fatal inflammation of the larynx. On inspection, it was ascertained that the acid had been surrounded by a cyst, formed by secretions from the stomach, which burst during the fit of coughing. This is an extraordinary case, and, so far as I know, unexampled in the history of poisoning. In explanation it has been suggested, that when concentrated sulphuric acid is dropped guttatim into albumen, a cyst of coagulum forms around the globules of acid, and preserves the remainder from its action. This effect, however, is only temporary, and it will not satisfactorily account for a large quantity of the poison being swallowed and retained in the human stomach for a fortnight without injury.

Among the *secondary* symptoms of poisoning by this acid, when the person survives some days or weeks, should be mentioned profuse salivation. This was observed in Mr. Tatham's case. (G. H. Rep. iv, 396, Oct. 1846.) Salivation commonly occurs about the second or third day —sometimes later. Desgranges observed a miliary eruption on the skin among the secondary consequences of poisoning by sulphuric and nitric acid. (Belloc, Cours de Méd. Lég. 120; Galtier, Traité de Toxicologie, i, 176.)

APPEARANCES AFTER DEATH.—Casper states from his observations, that the bodies of persons poisoned by sulphuric, and probably other mineral acids, resist putrefaction; they remain fresh for some time, and give out no offensive smell on inspection. He attributes this to the acid neutralizing the ammonia of the putrefactive process. (Handb. der Ger. Med. i, 400, 429.) The effects produced by this acid are not always found in the stomach; they may be confined to the region of the throat and air-passages. In an inspection of the body, the whole course of the alimentary canal from the mouth downwards, ought to be examined; since, in all recent or acute cases, it is in the gullet and *throat* that we obtain strong evidence of the action of a corrosive poison. The discovery of the usual marks of corrosion in these parts, is always highly corroborative of the signs of poisoning found in the stomach. During the inspection, the examiner must not omit to notice any spots on the skin produced by the action of the acid; these are commonly of a dark-brown color, and are situated about the mouth, lips, and neck. The appearances met with in the body will vary according to whether death has taken place rapidly or slowly. Supposing the case to have proved fatal very rapidly, the membrane lining the *mouth* may be found white, softened, and corroded; but this appearance may be absent. It was just now observed, that when the poison has been administered by a spoon, the mouth may escape the chemical action of the acid.

In the case of the Queen v. Thomas (Monmouth Lent Assizes, 1847), it was proved that the throat, gullet, and stomach of the deceased, an infant ten days old, were much corroded by sulphuric acid, which had been given to it in a diluted state; but there was no appearance of injury to the mouth. This was probably owing to a spoon having been used, and the poison having been poured down the throat slowly, as the mucous membrane was extensively corroded at the back part; and it was clear, therefore, that some corrosive substance had passed into the fauces. The mucous membrane of the throat and gullet will commonly be found corroded, having sometimes a brownish or ash-gray color. The corroded membrane of the gullet is occasionally disposed in longitudinal folds, portions of it being partly detached. The *stomach* if not perforated, is collapsed and contracted. On laying it open, the contents are commonly found of a dark brown or black color, and of a tarry consistency, being formed in great part of mucus and altered blood. The contents may or may not be acid, according to the time the patient has survived, and the treatment which has been adopted. On removing them, the stomach may be seen traversed by black lines, or the whole of the mucous membrane may be corrugated, and of a dark brown or black color. This blackness is not removed by washing. On stretching the stomach, traces of inflammation may be found between the folds, indicated by a deep crimson-red color. On removing the blackened membrane, the red color indicative of inflammation may be also seen in the parts beneath. Both the dark color and marks of inflammation are sometimes partial, being confined to insulated portions of the mucous membrane. When the stomach is perforated, the coats are softened, and the edge of the aperture is commonly black and irregular. In one case the fore part of the stomach presented a number of small holes, having black margins. In removing the stomach, the aperture is apt to be made larger by the mere weight of the organ. The contents do not always escape; but when this occurs, the surrounding parts are attacked by the poison. In a case which occurred at Guy's Hospital, the spleen, the liver, and the coats of the aorta, were found blackened and corroded by the acid, which had escaped through the perforation. The perforation of the stomach probably, in some instances, takes place after death from the chemical action of the acid. Dr. Craigie, of Edinburgh, thinks that even when there is no perforation of the stomach, the acid may find its way by transudation through the coats of the organ, in a very short time after it has been swallowed. In a case in which two ounces of the strong acid had been swallowed, and the person died in three hours and a half, he found that the peritoneum and the fluid contained in it, reddened litmus paper strongly. There was also a slightly acid reaction even in the serous membranes of the chest. It does not appear, however, that the nature of this acid was determined by the application of any test. When the person has survived for eighteen or twenty hours, traces of corrosion and inflammatory action may be found in the *small intestines*. In one case the mucous membrane of the ileum was corroded. In a case which occurred to Dr. Walker, of Inverness, a man died in twenty-five hours after he had swallowed two ounces of oil of vitriol. On inspection, the mucous membrane of the stomach was destroyed, and the whole surface darkened. The greatest amount of injury was at the intestinal end, where three small perforations were found. The orifice of the pylorus was swollen, constricted, and hardened; it was so small as to admit only of a silver probe. The duodenum had also much suffered. The first two inches of the arch of the aorta were very much inflamed. (Edin. Mon. Jour., June, 1850, p. 588.) Baron Dupuytren met with a case in which a woman died in seven hours from the effects of oil of vitriol. The mucous membrane of the gullet was raised by the action of the acid into longitudinal folds: that of the stomach was covered with black irregular spots—the coats were in a puffy state, and had a cauterized or burnt appearance. The pylorus and duodenum presented similar appearances. (Med. Gaz. vol. xi, 813.) In the case of a boy poisoned by concentrated sulphuric acid, the inner lining of the œsophagus was puckered, dry, and brittle: it was readily detached from the parts beneath, and came off in small scale-like portions. The stomach was not perforated, the coats were thin and allowed the contents to be seen through them. When opened, the whole of the mucous membrane was of a dark color, apparently stained by a bloody fluid, four ounces of which were contained in the stomach. The large end was unaltered, but the whole circumference of the smaller end almost midway between the two openings was black, irregular, rough, and thickened. The mucous membrane was here destroyed,—blood had been effused, and this had been coagulated and darkened by the action of the acid. The blood adhered to the corroded membrane. (Roupell on the Effects of Poisons, plate v.) The interior of the larynx as well as of the bronchial tubes, has also presented marks of the local action of the acid. The acid has thus destroyed life without reaching the stomach (ante, p. 228). It is important for the medical witness to bear in mind, that the throat and gullet are not always corroded; the mucous membrane sometimes presents merely black specks or points. Strange as it may appear, cases are recorded in which, notwithstanding the passage of the poison into the stomach, the gullet has escaped its chemical action. Mr. Dickinson has reported a case of poisoning by sulphuric acid in which there was no corrosion of the mouth or throat. The patient, a female, æt. 52, recovered in about five months. The stomach had probably sustained injury, as the most urgent symptoms were constant vomiting after taking food, and obstinate constipation. The quantity of acid swallowed was half an ounce, mixed with half an ounce of water. The patient felt immediately a burning sensation at the pit of the stomach (Lancet, Nov. 26, 1853, p. 502). The acid had here evidently lost its corrosive power by dilution. When the acid has been taken in a still more *diluted* state, the marks of inflammation on the mucous membrane are more decided, and the blackening is not so considerable. Nevertheless, the acid, unless too much diluted, acts upon and darkens the blood in the vessels, as well as that contained in the stomach, although it may not blacken the mucous membrane or the contents. Owing to the absence of corrosion in the throat and gullet, it might be assumed that sulphuric acid could not have been swallowed; and, in this respect, a case reported by M. Blondlot, of Nancy, is of importance. This gentleman was required to examine the clothes and viscera of an infant named Boullet, aged two months, that had died from the effects of sulphuric acid. The tongue, pharynx, and gullet presented no mark of corrosion, or any appearance indicating that a corrosive substance had been in contact with them. There was no eschar or alteration of color in any part. The appearances in the stomach were not very striking. An analysis showed that sulphuric acid existed abundantly on the clothing, but not a trace of the poison could be detected in the viscera. The case was remitted to MM. Devergie, Barse, and Lesueur, for examination; they confirmed the conclusions of M. Blondlot, and pronounced an opinion that, notwithstanding the absence of marks of corrosion in the viscera, and of the acid from their contents, the deceased had died from sulphuric acid administered to it. They were inclined to attribute the absence of the poison to vomiting and elimination by the urine. (Journal de Chimie Médicale, 1846, ii, 17.)

This case may be contrasted with that of Thomas, tried at the Monmouth Lent Assizes, 1847. The diseased infant, in this instance, was ten days old. The mother was charged with having administered sulphuric acid to it, and thus caused its death. The gullet, stomach, and intestines, were more or less corroded, but the *mouth* had escaped the action of the acid. No sulphuric acid was found in the body, but it was abundantly detected on the clothes of the child. The two cases are, therefore, very similar, and only differ in the fact, that there was evidence of corrosion by sulphuric acid in the English case. The counsel, who defended this case, contended that no "traces of unfairness" were to be discovered in the mouth; and how could a poison so strong and disagreeable to the taste, be got down the throat of an infant so young without leaving some ill effect behind in the mouth of the child." The learned judge (now deceased), who tried the case, is reported to have told the jury, that the evidence of medical witnesses was "generally a matter of conjecture or guess," and that the "traces of unfairness" (chemical corrosion) in the gullet and stomach "might have been produced by other substances (!) and not by the poison itself!" Hence, the reader will not be surprised to learn, that, in the case in which the evidence of death from sulphuric acid was strongest, the accused party was acquitted; while, in that in which it was less cogent (*i. e.* in the French case), but better sifted and appreciated, the prisoner was convicted and condemned to hard labor for life.

Dr. Chowne has reported a case which proves that a great legal mistake was here committed. A woman, æt. 52, swallowed from a cup about a tablespoonful of strong sulphuric acid. She informed Dr. Chowne that she was "strangled" the moment it got into her throat, and she fell to the ground. The usual symptoms appeared, and she died in two days. "The mucous membrane of the cheeks, gums, and tongue, was not excoriated at any part." (Lancet, July 10, 1847, p. 36.) It will be seen from this statement that even concentrated sulphuric acid may pass through the mouth without necessarily leaving any "traces of unfairness;" and yet the absence of these marks in Thomas's case was actually taken as a proof, not only that sulphuric acid (although diluted) had not been swallowed, but that the corrosion of the œsophagus, stomach, and intestines, depended on *natural* causes! The case related by Dr. Chowne establishes also another important fact, namely, that symptoms are immediately perceived, although the mouth may not be chemically corroded by the poison. The woman felt strangled so soon as the acid had reached her throat.

In a case, related at p. 230, in which the patient died on the third day, the appearances were peculiar. The mucous membrane of the mouth, tongue, epiglottis, and gullet, was of a yellow color,—the membrane could be easily peeled off. The parts about the larynx, where the acid had come in contact with them, were collored yellow and slightly swollen. The mucous membrane of the œsophagus was only superficially colored. The substance of the gullet was inflamed and swollen to thrice its natural thickness. The stomach contained about a pint of bright yellow fluid, which was not acid, and which did not yield any sulphuric acid on analysis. The greater end of the stomach presented the same yellow color as the gullet. The pyloric, or intestinal half, was blackened (quite charred), and raised into swollen masses or ridges. These would soon have sloughed off. The black appearance was owing to the carbonizing action of the acid on blood effused in the submucous tissue. The coats of the stomach, although not perforated at this part, were readily torn. The charred appearance of the stomach ended at the pylorus; but, for the first three inches, the folds (rugæ) of the duodenum (or small intestine) were slightly blackened. The remainder of the intestines presented no unnatural appearance. The small intestines contained a yellow fluid like that found in the stomach. The blood presented no remarkable appearance. The other organs were healthy.

When the poison has been taken in a *diluted* state, the marks of inflammation on the mucous membrane are more decided, and the charring is not so considerable. Nevertheless, the acid, unless too much diluted, acts upon and darkens the blood in the vessels, as well as that contained in the stomach, although it may exert no carbonizing action on the mucous membrane or on the contents.

Chronic poisoning.—The appearances just described will not, of course, be met with in protracted cases. If the person survive sufficiently long, all signs of inflammation and corrosion will disappear. Thus, in a case in which a child survived *twenty-five* days, the mucous membrane of the mouth and throat was sound but pale; that of the gullet, stomach, and duodenum was smooth, and equally free from any marks of corrosion or inflammation. (G. H. Rep. Oct. 1846, 396.) Casper met with two cases in children; in one the child survived three days, and in the other eight days. In both, the mucous membrane was pale. In the case of three days' duration there was no erosion of the gullet (Handb. der Ger. Med. i, 421).

The subjoined case shows the appearances which were met with when death did not occur until the eleventh day: C. D., a female lunatic patient, æt. 55, was admitted into Guy's Hospital on Oct. 5th, 1855, and died Oct. 16th. Two hours before admission, and before her breakfast, she drank a wineglassful of vitriol. On admission, no stains were perceived on the mouth, she was in a state of collapse, almost pulseless, the skin cold, and she was unable to swallow. In two hours she became warm, and vomited some thick bloody liquid, which contained sulphuric acid. There was also a discharge of blood from the bowels. In about ten or twelve hours she was able to swallow milk and arrowroot. She continued to vomit and pass blood by the bowels for several days. She was much reduced in strength, but there were no very urgent symptoms.

In four days after her admission she was able to swallow without difficulty. She had purging, but without blood, and the vomiting ceased. On the day of her death she sat up and spoke as usual, but in the evening was unexpectedly found dead.

The body was inspected seventeen hours after death. The mucous membrane of the mouth was white, of the throat and gullet pale, and covered by a granular deposit (of epithelium). The mucous membrane was not destroyed. The stomach was slightly contracted: it contained two ounces of yellow fluid, like the yolk of egg, and a thin membrane of the same color attached by one end. It consisted of the lining membrane of the upper part of the stomach, traversed by vessels filled with coagulated blood. The central portion of the organ had also lost its mucous membrane. The destruction of the membrane continued into the duodenum. The small intestines were congested, and a false membrane was found in the jejunum. The whole of the large intestines was acutely inflamed, the interior being covered by adherent false membranes. The liver and kidneys were healthy. In a case which proved fatal on the sixteenth day, the stomach was found perforated, but adherent to the coverings of the abdomen. The mouth, throat, and gullet presented no marks of corrosion. The quantity of acid taken (consisting of two-fifths of strong acid) amounted to two ounces. When first seen, the surface of the mouth was white. (Med. Times and Gazette, Dec. 19, 1857, p. 629.)

In other instances the mucous membrane has been found either entirely destroyed, or more or less ulcerated. M. Louis met with a case fatal after two months. The patient suffered under the usual primary symptoms. During the latter part of the illness there was habitual constipation, with great thirst. Three days before death, black liquid matter, probably altered blood, was passed from the bowels. On inspection, the mucous membrane of the gullet was destroyed; it was ulcerated, and the first portion perforated. In some spots the muscular coat was wholly destroyed, while in others it was generally thickened. The mucous membrane of the stomach was found ulcerated, and in some parts gelatinized; at the lower part it had a bluish tint (Med. Gaz. vol. xiv, p. 31). Similar ulcerations in the gullet and stomach were found in the case of a female who died in sixteen days, from the effects of an ounce and a half of sulphuric acid. Dr. Clendinning, who reports the case, states that there was ulceration and loss of substance at the two ends of the stomach, with thickening and corrugation at the apertures. The pylorus was thickened and narrowed. The stomach was much injected and swollen, and at the greater end there was softening and erosion. Obstinate constipation, with great difficulty in swallowing, were among the most urgent symptoms (Med. Gaz. vol. xvii, p. 340). This destruction of the inner coat of the stomach leads to death, by impairing the function of digestion. In several cases, the aperture of the pylorus has been found much contracted. (Galtier, op. cit. i, 197.) Sometimes stricture of the œsophagus is a consequence of the local action of the acid. (A rare case of recovery from stricture thus produced, is reported in Med. Times and Gazette, May 15, 1858, p. 510.) The common secondary causes of death in these chronic cases, are fever, irritation, or exhaustion. There will be occasionally a difficulty in connecting death with the poison when the person survives; but it appears to me the difficulty was unnecessarily created in the following case (Reg. v. Connell, Central Criminal Court, Nov. 1852). The prisoner had administered oil of vitriol to her mistress's infant, seven weeks old. The child recovered from the first effects, but was subsequently attacked with inflammation of the windpipe. It died in convulsions on the sixteenth day from the date of taking the poison. The medical witness stated, that owing to the effects of the acid, the child was unable to take food; this brought on great debility, and this debility was likely to induce convulsions, for which no other cause was apparent. Convulsions, however, were here treated, not as a symptom, but as a specific disease. The medical witness could not say, in cross-examination, that they might not have proceeded from other causes, and on this wide contingency, the jury acquitted the prisoner (see ante, p. 142, CONVULSIONS). The prisoner was subsequently indicted for the felonious administration of the oil of vitriol. A bottle was traced to her possession, which she said (being a Roman Catholic) contained "holy water." On analysis, however, the acid which had corroded the skin and dress of the child, was found in it. For the defence it was suggested that the "unfortunate occurrence" was the result of accident, a theory which the jury adopted! The case of Reg. v. Brennan, for poisoning an infant with oil of vitriol (Chester Win. Ass. Dec. 1856), ended also in a verdict of not guilty, although neither accident nor suicide could be suggested.

Absorption and Elimination.—It has been a disputed question, whether sulphuric acid is or is not absorbed and carried into the circulation in cases of acute poisoning. M. Bouchardat considers that it is absorbed, and that it causes death by leading to a coagulation of the blood in the heart, aorta, and large bloodvessels. He has found coagula (clots of blood) in two cases in considerable quantity; and in one of them, the lining membrane of the aorta was reddened. (Annales d'Hygiène, 1837, i, 362.) I have observed this last-mentioned appearance in one case, as well as the occurrence of coagula in two instances; but there does not seem to be any reason for believing that they result from the action of a portion of sulphuric acid absorbed. (Galtier, Toxicologie, i, 190, 191.) In analyzing these coagula taken from persons who had been killed by sulphuric acid, I have not found a trace of the acid in them. According to Orfila, the absorption of these mineral acids may take place owing to their compounds with albumen being soluble and neutral. There is no doubt that all these albuminous compounds are soluble in a large quantity of water, but they are insoluble when much acid is present. In a case reported by Dr. Letheby to the Pathological Society, a chemical analysis of the urine proved that the acid was rapidly eliminated by this secretion. The quantity thus passed within four days was considerable. (Med. Gaz. xxxix, 116; ante, p. 41.)

The reader will find that this subject has been fully considered by Dr. Geoghegan, to whose experiments reference has been elsewhere made (ante, p. 41; also Med. Gazette, vol. xlviii, p. 330). In a case in which a woman swallowed one ounce and a half of oil of vitriol, and survived thirty-one hours, he states that he found traces of sulphuric acid in the serum of the pericardium and the kidney. There was none in the blood; but a quantity of free phosphoric acid, which he considered to be the equivalent of the sulphuric acid which had been absorbed and had decomposed the alkaline phosphate of the blood; the alkaline sulphate produced having been eliminated from the kidney. The stomach was perforated, and the organ was empty. In Dr. Walker's case (ante, p. 232), we are informed that there was a trace of sulphuric acid in the serous fluid at the base of the brain, and a larger quantity in the blood contained in the heart. There was none in the stomach, and only a slight trace in the duodenum. (Ed. Monthly Journ. June, 1850, p. 538.)

It will be apparent from these statements, that the results of experiments for the detection of absorbed sulphuric acid in the blood are conflicting. According to Casper, in this form of poisoning the blood has always an acid reaction, even in those organs which are healthy; it has also the consistency of treacle and a cherry-red color. He found, in one instance, the pericardial fluid acid, and in another,—the case of a pregnant female,—the amniotic fluid was acid (Handbuch der Ger. Medicin, 1857, vol. i, p. 400, 430); but he did not test the liquid for sulphuric or phosphoric acid. Professor Carus has reported a case in which the acid was taken by a pregnant female, and it was found not only in the water of the amnios, but in the cavity of the pleura and peritoneum of the fœtus, as well as in the heart and bladder. (Beck's Med. Jur. vol. ii, p. 429; and Bulletin des Sciences Médicales, vol. xiii, p. 72.)

FATAL DOSE.—The dangerous effects of this poison appear to arise more from its degree of concentration, than from the absolute quantity taken. The quantity actually required to prove fatal, must depend on many circumstances. If the stomach is full when the acid is swallowed, its action may be spent on the food and not on the stomach; and a larger quantity might thus be taken than would suffice to destroy life if the organ were empty. In one case, one drachm of sulphuric acid destroyed life in seven days:—in another (Humphrey's case, ante, p. 113, also Med. Gaz. viii, 77), about one drachm and a half destroyed life in two days. In Mr. Schwabe's case, six drachms destroyed life in twenty-four hours. (Med. Gaz. xxxvi, 826.) In one instance, a patient survived fifty-five hours after taking three fluid-ounces of the concentrated acid (Dr. Sinclair, Med. Gaz. vol. viii, p. 624); in another, related by Sobernheim, a man swallowed an ounce and a half of the concentrated acid, and yet slowly recovered from its effects. (Handbuch der Prakt. Tox. 684.) In a case, quoted by Dr. Craigie, a young woman, aged 18, recovered after having taken *two ounces* of concentrated sulphuric acid. She was completely restored in about eighteen days. (Ed. Med. and Surg. Jour. April, 1840.) Another instance of recovery after two ounces of the concentrated acid had been taken, is reported by Mr. Orr (Med. Gaz. iii, p. 255). A remarkable instance of recovery from a large dose was observed in a case which occurred in the practice of M. Biett. The patient, a man, aged 31, swallowed by mistake *three ounces* (by weight?) of commercial sulphuric acid. Severe burning pain and vomiting immediately followed; the man fell and rolled on the ground in agony, but nevertheless was able to walk some distance to the hospital without assistance, although he rested occasionally. Milk and magnesia were freely given to him, and in a week he perfectly recovered. The most striking symptom was excessive salivation, which set in on the second day and continued for three days. (Galtier, Toxicologie, i, 186.) It is probable that, in these instances of recovery from large doses, the greater part of the poison is expelled in the matter first vomited. In Dr. Letheby's case, the patient, a child only nine years old, recovered in a short time, after having swallowed one ounce of concentrated sulphuric acid. In this case nothing was done for five minutes. For the first few days the patient was copiously salivated. (Med. Gaz. xxxix, 116.) The smallest quantity which I have been able to meet with as having proved fatal, was in a case already quoted. Half a teaspoonful of concentrated sulphuric acid was given to a child, about a year old, by mistake for castor oil. The usual symptoms came on, with great disturbance of the respiratory functions; and the child died in twenty-four hours. The quantity here taken could not have exceeded *forty drops.* (Med. Gaz. xxxix, 147; see also ante, p. 181.) It is, however, doubtful whether this small quantity would have proved fatal to an adult. The smallest fatal dose which Dr. Christison states he has found recorded, was *one drachm;* it was taken by mistake, by a stout young man, and killed him in seven days. (Op. cit. 162.)

Period at which death takes place.—It has been already stated, that the average period at which death takes place in cases of acute poisoning by sulphuric acid, is from eighteen to twenty-four hours. Death has occurred suddenly and unexpectedly, when the patient appeared to be progressing to recovery. If the stomach is perforated by the acid, it proves more speedily fatal. In an instance, reported by Dr. Sinclair, in which a child about four years old died in four hours,—the stomach was perforated. When the acid acts upon the air-passages, death may be a still more speedy consequence from suffocation; and owing to this, it appears to be more rapidly fatal to children than adults. But in one case of an adult, elsewhere recorded (ante, p. 229), it must have destroyed life by its action on the air-passages during swallowing. Dr. Craigie mentions a case in which three ounces of concentrated sulphuric acid destroyed life in three hours and a half; but the shortest case on record is, perhaps, that mentioned by Remer in Hufeland's Journal. In this instance death took place in two hours. A case, which proved fatal in two hours, is reported by Casper (Handb. der Ger. Med. 1857, i, 422). The stomach in this case was quite blackened, and so soft that it gave way like blotting-paper. The stomach was found perforated, and the omentum blackened. There had been some vomiting, but sulphuric acid was detected in the corroded parts. In the case of a child a year and a half old, criminally poisoned by its mother, death took place in *one hour.* There were parchment-colored streaks and spots at the corners of the mouth, and on the arms and hands of the child. The stomach contained a dark acid fluid, consisting of blood and mucus,—the coats were softened to solution. The gullet was firm, and the mucous membrane had a gray color. (Op. cit. p. 426.) A case, in which life was destroyed with equal rapidity, is reported in the Edinburgh Monthly Journal, 1854 (p. 138). A woman took a wine-glassful of oil of vitriol (s. g. 1·833), in mistake for ale. In spite of treatment, she died in one hour. The stomach was perforated, and the acid had escaped; the coats were softened; the edges of the aperture were ragged; and the mucous surface, generally, was mottled with dark-brown patches. Another instance of death in two hours is quoted by Galtier (Toxicologie i, 193). A case is reported by Mr. Watson, in which a woman swallowed two ounces of the strong acid. She died in *half an hour*, but it appears that a quarter of an hour before death she had made a deep wound in her throat, which gave rise to great hemorrhage. The stomach was found extensively perforated; but it is highly probable that in this case the wound accelerated death.

On the other hand, there are numerous instances reported in which the poison proved fatal from secondary causes, at periods varying from one week to many months. In one instance, a child recovered under treatment from the first effect, but died of starvation after twenty-five days, from the impossibility of retaining any kind of food on its stomach. (G. H. Rep. Oct. 1846, 396.) A remarkable case of a similar kind occurred to Dr. Wilson of the Middlesex Hospital, and is referred to by Mayo in the Outlines of Pathology. A young woman swallowed about a tablespoonful of sulphuric acid on the 4th of January, and died from its effects on the gullet, on the 14th of November following. She gradually wasted away, and sank from innutrition. This was forty-five weeks, or *eleven months*, after she had swallowed the poison. There is no doubt that the acid may prove fatal at all intermediate periods, and at intervals much longer than this; but the longer this event is protracted, the more difficult will it be to ascribe death to its effects. Dr. Beck refers to a case in which death took place from stricture of the œsophagus *two years* after the poison had been taken. (Med. Jur. vol. ii, p. 426.)

Treatment.—Although it is the general practice to give magnesia and chalk freely in milk or water, it appears to me, from a case which I had the opportunity of examining, that a solution of carbonate of soda in milk and water, properly diluted, and given in small quantities at intervals, would act more effectually and more speedily in neutralizing the poison. The insoluble particles of magnesia adhere closely to the mucous membrane, and do not readily come into contact with the acid. In examining the dark tarry matter vomited by a child half an hour after the concentrated acid had been taken, I found it still intensely acid, although during the whole period, a magnesia mixture had been freely given in divided doses. This objection would not apply to the use of bicarbonate of magnesia or lime; and the evolution of carbonic acid would be a minor evil compared with the action of sulphuric acid in an unneutralized or imperfectly neutralized condition. Sobernheim and Simon relate several instances in which persons who had taken this poison, were apparently saved by the free use of these alkaline diluents. In the absence of these substances, oil may be freely administered. Carbonate of magnesia mixed with oil has been sometimes beneficially given. There is often great difficulty in making the patient swallow; the throat being swollen, and blocked up with shreds of tough coagulated mucus and phlegm. Hence it has been recommended to employ the stomach-pump for the purpose of injecting the liquids into the stomach. The use of this instrument ought, however, if possible, to be avoided; since it is only likely to lacerate and perforate the structures which are softened and corroded by the acid. When there are symptoms of suffocation from an affection of the larynx, tracheotomy must be resorted to. On the whole, the antidotal treatment of cases of poisoning by sulphuric acid has not been very successful, the patient not having been seen sufficiently early by a medical man to give much hope of success. It should be remembered, that the poison begins to act instantly; and if the stomach be at the time empty, there is but little prospect of saving the patient. These cases often prove fatal even when every trace of the poison has been removed from the stomach, owing to the extensive local injuries produced.

That this antidotal treatment may, however, occasionally be the means of saving life, the following case, related by Barzellotti, will show. A man, aged 40, swallowed by mistake a quantity of the oil of vitriol, and was brought to the hospital of Santa Maria Nuova. He was suffering from intense burning pain in the throat and abdomen, as well as from other severe symptoms; calcined magnesia in water was given to him at short intervals, until it was supposed enough had been taken to neutralize the acid. A quantity of tepid water was then administered to promote vomiting; and, on examining the vomited matters, it was found that the sulphuric acid was neutralized by magnesia. The patient was then bled; leeches and fomentations were applied to the stomach, and bland liquids prescribed. The man slowly recovered, suffering from difficulty of swallowing and severe cough. In one fit of coughing he expelled a mass of false membrane, of the form and size of the gullet. The abdomen and throat were tender at the time of his discharge. (Questioni di Medicina Legale, ii, 307.) It is to be observed, that cases of accidental poisoning like this, much more frequently do well than those of suicide—the quantity of poison swallowed being in general small. A case reported by Dr. Borgstedt of Minden, shows the best mode of after-treatment. A boy, aged three years, swallowed from one to two drachms of oil of vitriol. Some train-oil was given to him. When medical assistance was procured, the patient was lying speechless and motionless on his back, face pale, eyes deeply sunk and closed, breathing difficult, and accompanied by a rattle. The skin around the mouth, as well as the lining membrane, had been destroyed by the acid. Carbonate of potash was given at intervals, and this was followed by the vomiting of a dark-brown slimy matter. When vomiting had ceased, an oily emulsion with carbonate of magnesia, was given every half-hour. Some febrile symptoms set in, with severe pain in the region of the stomach. Leeches were applied, and repeated for three days. The eschars from the destroyed skin and membrane were smeared with olive oil and yolk of egg, with great relief to the patient. The only nourishment allowed was milk. The fever and abdominal tenderness disappeared about the fourteenth day. On the first day the motions had a natural color. From the second to the seventh day they were very hard, and appeared like slaked lime; from the seventh to the thirteenth day they assumed an ash-gray color; and on the fourteenth day they had their usual characters. (Casper's Wochenschrift, May 9, 1846.)

The following case of successful treatment, which was reported by Mr. Gardner to the Lancet (August 25, 1838), deserves to be here mentioned. A young man swallowed half an ounce of strong sulphuric acid. The usual symptoms appeared; milk and carbonate of magnesia were freely given. This person recovered in twelve days. One of the secondary symptoms was profuse salivation.

It is worthy of remark, that several cases of recovery have taken place, in which no chemical antidotes had been administered. The treatment consisted simply in giving large quantities of gruel and milk; and there is no doubt, that any thick viscid liquid of this description, as, for example, linseed oil and lime-water, or flour and water, must be beneficial, by combining with the acid and arresting its corrosive effects. In short, such a liquid would act much in the same manner as the presence of a large quantity of food is known to act, when the acid is swallowed soon after a meal. In all cases, it would be advisable to combine the use of chemical antidotes with the copious administration of mucilaginous drinks.

CHEMICAL ANALYSIS.

This acid may be met with either concentrated or diluted; and a medical jurist may have to examine it under three conditions: 1. In its simple state. 2. When mixed with organic matters, as with liquid articles of food or in the contents of the stomach. 3. On solid organic substances, as where the acid has been thrown or spilled on articles of dress or clothing.

In the simple state.—If *concentrated* (oil of vitriol), the acid possesses these properties: 1. A piece of wood, or other organic matter, plunged into it, is immediately carbonized or charred. 2. When boiled with wood, copper cuttings, or mercury, it evolves fumes of sulphurous acid; this is known by the odor, as well as by the acid vapor first rendering blue, and then bleaching starch-paper dipped in a solution of iodic acid. 3. When mixed with its volume of water, great heat is evolved (nearly 200° F. in a cold vessel). In this state the acid does not carbonize wood and is not decomposed when boiled with copper cuttings.

The Diluted Acid.—For the acid in the *diluted* state, but one test need to be applied: a solution of a salt of baryta—the *Nitrate of baryta* or the *Chloride of barium.* Having ascertained by test paper, that the liquid is acid, we add to a portion of it a few drops of nitric acid, and then a solution of nitrate of baryta. If sulphuric acid be present, a dense white precipitate of sulphate of baryta will fall down,—which is insoluble in all acids and alkalies. If this precipitate be collected, dried, and heated to full redness in a small platina crucible, with two parts of vegetable charcoal powder, it will, if a sulphate, be converted to sulphuret of barium. To prove this, we add to the calcined residue, diluted muriatic acid, at the same time suspending over it a slip of filtering paper moistened with a solution of acetate of lead; or, we place the residue on a slip of glazed card (coated with carbonate of lead), scraped and wetted on the surface. (The card should be first tested for lead: because some kinds

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of glazed cards are made without lead.) If the original precipitate were a sulphate, the vapor now evolved will be sulphuretted hydrogen, known by its odor, and by its turning the salt of lead or staining the card of a brown color. Instead of charcoal, we may use an equal bulk of cyanide of potassium as the reducing agent, and the experiment may then be performed in a small reduction tube over a spirit lamp. On breaking the tube and placing the powder on a glazed card (containing lead), previously wetted, the stain of sulphuret of lead will be perceived;—or, the calcined residue may be dissolved in water and tested. The smallest visible quantity of sulphate of baryta thus admits of easy detection.

The delicate action of this test is such, that a solution containing not more than the 1-25,000th part, by weight, of sulphuric acid, is precipitated by it. When the sulphuric acid is diffused through a minimum of water, the barytic test gives a perceptible precipitate with the 1-110th part of a grain of the acid. If, however, this small quantity be diluted with an ounce of water, the test produces no perceptible change. In these experiments distilled water must be used, since all kinds of river and spring water are precipitated by the test. With regard to the reduction of the precipitate to the state of sulphuret by charcoal or cyanide of potassium, I have found that one-half grain of the sulphate of baryta will yield satisfactory evidence; and a quarter of a grain will give traces of sulphur, although somewhat indistinct. This is equivalent to about one-eighth of a grain of common oil of vitriol (bihydrate). In cases of poisoning, however, we either find the acid in larger proportion, or it is altogether absent. Orfila recommends that the diluted acid should be concentrated by evaporation and then treated with metallic copper, to liberate sulphurous acid; but this process is more troublesome, and less likely to prove satisfactory than that just described. Animal charcoal should not be used in this analysis, as this frequently contains traces of a sulphuret.

Objections to the process.—Provided the process be carried out with pure materials to the production of *sulphuret* of barium,—it is not open to any objection. There are some points, however, which require consideration: 1. A solution of alum, of any acid *sulphate*, or of *bisulphate of potash*, might be erroneously pronounced to be free sulphuric acid; for alum and the acid sulphates would, with the tests, give all the reactions which have been here described. The answer to this objection is very simple: we must slowly evaporate a portion of the suspected liquid in a watch-glass,—there will be a saline residue if there be a solution of alum, or any salt,—otherwise not; for sulphuric acid should be entirely dissipated by heat, or it should leave only the faintest trace of sulphate of lead. 2. The quantity of free sulphuric acid present might be erroneously estimated, in consequence of some simple medicinal sulphate (as Epsom salt) being mixed with it. This may be determined also by evaporation; and the free sulphuric acid separated by warming the liquid, and adding finely-powdered carbonate of baryta, until effervescence ceases. The precipitate formed would be sulphate of baryta, and represent the free sulphuric acid present.

There is, however, another source of error: any acid mixed with a common sulphate employed in medicine, might be mistaken for free sulphuric acid; as, for example, a mixture of citric or acetic acid or vinegar with sulphate of magnesia. This may be suspected when any saline residue is left on evaporating the mixture. In such a case carbonate of baryta would not separate the free acid. It is easy to procure by evaporation and incineration the sulphate from a given measure of the liquid, and we can then determine whether the sulphate of baryta obtained is greater than, or equal to the weight of alkaline sulphate present.

In liquids containing organic matter.—If sulphuric acid be mixed with such liquids as porter, coffee, or tea, the process for its detection is substantially the same, the liquid being rendered clear by filtration previously to adding the test. The sulphate of baryta, if mixed with organic matter, may be purified by boiling it in strong nitric acid; but this is not commonly necessary, as the reduction of the precipitate may be equally well performed with the impure, as with the pure sulphate. Some liquids generally contain either sulphuric acid or a sulphate, such as vinegar and porter, but the acid is in very minute proportion: therefore, if there be an abundant precipitate, there can be no doubt, *cæteris paribus*, that free sulphuric acid has been added to them. Should the liquid be thick and viscid like gruel, it may be diluted with water, and then boiled with the addition of a little acetic acid. For the action of the test, it is not necessary that the liquid should be absolutely clear, provided it be not so thick as to interfere mechanically with the precipitation of the sulphate of baryta. So far with regard to articles administered, or of which the administration has been attempted.

Vomited matters.—These will commonly be found highly acid, reddening litmus paper, and causing effervescence with carbonated alkalies; they may be diluted with water, boiled, filtered, and tested in the manner above described. The acid is sometimes so intimately combined with decomposed mucus and blood, that it requires long boiling in order to separate it. If the patient has been under treatment, the matters obtained from the stomach may have *no acid* reaction, either from the copious administration of water and abundant vomiting, or from an antidote having been used, such as soda or magnesia. If on adding the test to a *neutral* liquid, there is a precipitate, sulphuric acid can be present only in the state of *sulphate*. If this precipitate be abundant, it cannot be due to the presence of minute traces of sulphates in the gastric and salivary secretions; but still it would be improper to infer from this chemical fact alone, that sulphuric acid had been swallowed, because it is well known that some saline sulphates, such as those of magnesia and soda, are often exhibited in large quantities medicinally, and it might be fairly objected to this evidence, that the precipitate was due to the presence of one of these salts. The symptoms, as well as other circumstances, would here aid the witness in forming an opinion,—chemistry alone might mislead him.

In examining an organic liquid which has no acid reaction, it must be remembered that there are many salts in common use, some of them being medicines, which precipitate the barytic test. These are—all the soluble carbonates, iodates, phosphates, borates, tartrates, and oxalates. It is to be observed, however, that not one of these substances is precipitated by the test, provided the liquid for analysis be much diluted and acidulated with nitric acid before adding it. Should nitric acid alone produce any turbidness in an organic liquid, this may be again boiled and filtered before it is tested.

Contents of the Stomach.—If the patient survives, the analysis will of course be confined to the matters vomited. When the case proves fatal, we may, however, be required to examine the contents of the stomach. Should these be acid and give a precipitate with the test, it may be said that the acidity was due to the acids naturally contained in the gastric secretions (the muriatic and acetic), which, however, are in small proportion; or to some acid liquid, taken in the form of medicine or otherwise before death; the precipitation by the test might also be ascribed to the presence of some medicinal sulphate. If the contents were not acid, then the effect produced by the test might be ascribed to the latter circumstance alone. Objections of this kind are at once removed not merely by resorting to the processes above described, but by noting particularly the presence or absence of the usual changes produced by mineral acids in the throat, gullet, and stomach. A chemist might decide from an analysis alone; but a medical jurist should take into consideration the symptoms under which the deceased labored, and the appearances found in the body, before he pronounces an opinion from the results of his experiments.

Supposing the contents to give no evidence, or but slight evidence, of the presence of the acid, we must then boil the altered or decomposed portions of the stomach in water for an hour, filter, and apply the tests to the filtered liquid. But still no evidence of the presence of the acid may be obtained. Under these circumstances, it has been proposed by MM. Taufflieb, Devergie, and others, to heat the stomach to a high temperature in a retort, the beak of which is plunged into a mixture of iodic acid and starch. (Ann. d'Hygiène, 1835, 1427.) It is assumed that the non-discovery of the acid is due to its combination with the substance of the stomach in a form so intimate, that water cannot separate it. The application of heat therefore would, in the process above mentioned, lead to a decomposition of the sulphuric acid by the carbon of the animal matter, and its transformation to sulphurous acid. This would be immediately indicated by the production of the blue iodide of farina in the receiver. This process is objectionable, because mucous membrane and animal substances contain sulphur, which is evolved in combination at a high temperature, and the products would affect the test as if sulphuric acid were present. (Med. Gaz. vol. xxxvii, p. 954.) It has been suggested that alcohol or ether might be used for separating sulphuric acid from an alkaline sulphate when there is a mixture of these in the contents of the stomach. Alcohol, however, takes a portion of acid from the acid sulphates, and ether will dissolve diluted sulphuric acid only to a very limited extent. The presence or absence of alkaline sulphates, when any doubt exists, should be determined by incineration. Traces of sulphates will be found in animal liquids as well as in the incinerated residue of the stomach and bowels (Galtier, Toxicologie, i, 173). The condition of the stomach and gullet will show whether sulphuric acid, as such, has been in contact with these parts.

It is a medico-legal fact of considerable importance, that the contents of a stomach in a case of poisoning by sulphuric acid, are sometimes entirely free from any traces of this poison, even when it has been swallowed in large quantity. The acid is not commonly found when the individual has been under treatment,—when there has been considerable vomiting, aided by the drinking of water or other simple liquids, or when the person has survived several days. If the case has been under treatment, the acid is either wholly absent or neutralized by antidotes. A girl swallowed four or five ounces of diluted vitriol, and died in eighteen hours. No portion of the acid could be detected in the stomach; but she had vomited considerably, and the acid was easily proved to exist in the vomited matters, by examining a portion of the sheet of a bed which had become wetted by them. In another case, nearly two ounces of the concentrated acid were swallowed; the patient died in twenty-five hours; the stomach was most extensively acted on, and yet no trace of the acid could be discovered in the contents. The liquidity of the poison, and the facility with which it becomes mixed with other liquids and ejected by vomiting, will readily furnish an explanation of this fact. In many cases of poisoning by sulphuric acid, therefore, a medical witness must be prepared to find, that chemical analysis will furnish only negative results. This, however, is not inconsistent with death having taken place from the poison. The facts are so conclusive on this point, that I should not have thought it necessary to add to the evidence accumulated on the subject, but that the erroneous statement has recently been put prominently before the public, and, to a certain extent, received on the oaths of "experts," that no person can die from poison except the poison be found in the body (see ante, p. 170). Casper has dealt with this question. He relates three cases of poisoning by sulphuric acid, which occurred to himself, one which proved fatal in eight days, a second in five days, and a third in three days. In not one instance could a trace of the poison be found. (Handb. der Ger. Med. i, p. 421, 429.) In the second case two tablespoonfuls were swallowed by a girl. The analysis revealed merely the accidental presence of a fractional part of a grain of alkaline sulphate in the stomach and bowels. Thus there was an entire failure of proof from chemistry, while the facts of the case, and the appearances in the body, established conclusively that death had really been caused by sulphuric acid. In a recent instance, in which death took place on the eleventh day, I found no trace of sulphuric acid in the body. If the stomach should be perforated, the contents will be found in the abdomen, or perhaps in the lower part of the cavity of the pelvis: they may then be collected, boiled with distilled water, and the solution examined for the acid by the process already described. If the contents of the stomach are highly putrefied, the sulphuric acid may be found combined with ammonia.

On solid organic substances.—In cases of poisoning, sulphuric acid is sometimes spilled upon articles of clothing, such as cloth or linen, and here a medical jurist may succeed in detecting it, when every other source of chemical evidence fails. (Case of Humphreys, ante, p. 113.) Again, sulphuric acid is often used maliciously, by throwing it on the person, an offence which, when accompanied with bodily injury, renders the offender liable to a severe punishment. On such occasions, proof of the nature of the corrosive liquid is required; and this is easily obtained by a chemical examination of parts of the dress. The process of analysis is very simple. The piece of cloth should be digested in a small quantity of distilled water at a gentle heat, whereby a brownish-colored liquid is commonly obtained on filtration. If sulphuric acid be present, the liquid will have an acid reaction, and produce the usual effects with the barytic test.

Spots on clothing, if produced by the concentrated acid, are known in general, 1. By the black woollen cloth having its color changed to a dirty brown, and the spot acquiring a red border after a few days. Diluted sulphuric acid produces at once, on black cloth, a red stain, which slowly becomes brown. 2. By their remaining damp or humid for a considerable period,—the sulphuric acid being a very fixed acid, and readily absorbing water. That no objection may be offered to the result of an experiment of this kind, it is necessary that another part of the dress should be tested, in order to show that the sulphuric acid detected is not due to the presence of any sulphate in the dress. Many articles of clothing, it must be remembered, yield slight traces of sulphates, but not of sulphuric acid, when boiled in water. In the attempted erasure of writing from paper by diluted sulphuric acid, the same process will detect its presence. All white organic substances, such as calico or paper, although not blackened by diluted sulphuric acid, become, when impregnated with it, immediately charred on exposure to a moderate heat. The fibre of linen or cotton is slowly destroyed, even when the sulphuric acid forms only 1-30th part of the liquid. It may be objected to the medical evidence that this acid is used in bleaching cloth (Queen v. Thomas, Monmouth Lent Ass. 1847); but a medical witness must bear in mind, that the cloth is also passed through a bath of chloride of lime and of alkali, so that all traces of free sulphuric acid are thereby removed. The color of black leather is not changed by sulphuric acid. One article of clothing may be impregnated with the acid by imbibition through another article which covers it. In Reg. v. Brennan (Chester Winter Ass. 1856), it was proved that the frock of an infant was stained with sulphuric acid. On evidence being given that the child wore a pinafore over the frock, it was at once assumed, by the learned counsel, that the frock could not have been stained through the pinafore. This was made a point in the defence; but in a scientific view the fact could create no difficulty whatever, for the acid may penetrate several layers of clothing in a degree sufficient to be detected by chemical analysis.

It is proper in these cases to prove that the stain has not been caused by an acid sulphate, such as bisulphate of potash. A solution of this substance gives to black cloth a reddish stain, which resembles that produced by diluted sulphuric acid. The salt may be found in the stuff by incineration.

In dyed articles of linen and cotton, the effect of the acid varies with the dye. In all cases the organic matter is sooner or later corroded and destroyed both by the concentrated and the weak acid. If the dress be dyed blue by indigo, the color will be unchanged. (Nitric acid discharges the color of indigo, turning it yellow.) If dyed with archil and some other blues, the spot may be reddened, and red streaks will be found wherever the acid vomited matter has passed over the dress. Logwood and madder, which are largely used in dyeing many of the common calicoes, are turned of a yellow color by strong acids; and wherever an iron mordant has been employed in the pattern, there will be a rusty red spot. In a very diluted state the acid is slow in acting, and the stain when recent is red. I have found by experiment, that whether the acid is used in a pure state, or darkened by organic matter, the effect is the same; and it would be impossible to say whether the colorless or colored carbonized acid had been employed for the purpose of producing it. These stains on the dress in cases of imputed poisoning, require a close chemical examination.

I have ascertained by experiment, that sulphuric acid may be easily detected on articles of clothing after many years' exposure. In January, 1831, a small quantity of this acid was spilled on a black cloth dress; it had been exposed in an open jar to the air for the long period of *twenty-seven years*. In 1858, the cloth was soft, corroded, and of a deep brownish-black color; it yielded to water a brownish-colored acid liquid, in which sulphuric acid was easily detected. In a case of poisoning which occurred in 1832, the acid was partly spilled on a dress of printed cotton. This had been likewise exposed for twenty-six years; the organic fibre was completely corroded by the acid, and reduced to a kind of humid powder; from this substance, by the addition of water, a liquid was obtained, the acidity of which was proved by the barytic test to be due to the presence of sulphuric acid. These facts are of some interest, because it has been generally supposed that the stains on clothes lose all traces of the acid, partly by decomposition in contact with organic matter, and partly by evaporation; but it is hereby evident, that such stains, if not removed by washing, may be, in some instances, detected for a period of time much longer than is ever likely to be required in any medicó-legal investigation.

In determining the presence of sulphuric acid in clothing composed of vegetable fibre, I have found a modification of the iodic acid test extremely delicate and easy of application. It may be thus applied: About half a grain of the article of dress (cotton) (impregnated with sulphuric acid) is introduced into a short piece of glass tube, about one-eighth of an inch in diameter, and closed at one end. The stuff is then gently heated, and at the same time a piece of paper, previously saturated with starch and moistened with a drop of iodic acid, is brought near to the mouth of the tube. The blue iodide of farina is immediately produced by the sulphurous acid formed at the expense of the sulphuric acid, locked up in the organic substance. This process is only strictly applicable to the examination of those articles of dress which contain *no sulphur*, as cotton or linen, or which are not impregnated with compounds containing sulphur; thus the presence of serum, blood, or mucus, owing to these substances containing sulphur, would lead to fallacious results. All kinds of flannel and woollen cloth contain sulphur as a natural constituent; hence, whether sulphuric acid be present or not, they will decompose iodic acid by heat. It is highly necessary to bear in mind this fact, as the following case will show. In April, 1846, Mr. Eastes, of Folkestone, brought to me for examination the stomach of a man who, it was supposed, had been poisoned by sulphuric acid. There was a dark coagulum of blood and mucus lining the stomach, and the surface of the mucous membrane was reddened; but the most careful analysis showed that not a particle of sulphuric acid was present. A portion of the string with which the stomach was tied (which was of a dark color) was dried and heated in a tube. The vapor immediately decomposed iodic acid, which I attributed to its having been impregnated with part of the dark coagulum of blood and mucus, as no sulphuric acid was found in it, and an unstained portion of string produced no effect on iodic acid. (Med. Gaz. xxxvii, 954.) In order to obviate any fallacy in the employment of this test, the analyst should always examine an unstained portion of the stuff, whether linen or cotton. We cannot trust to this process, in the analysis of any kind of woollen cloth or silk. In boiling some articles of clothing in water, an acid liquid may be obtained presenting the properties of sulphuric acid. In a case tried at the Central Criminal Court, 1840, Dr. R. D. Thomson found that the material of a stained hat, probably owing to the presence of alum and copperas, gave traces of sulphuric acid even in the portion which was uninjured. By a comparative quantitative analysis, he found a larger quantity of acid in the stained portion. Still the result shows that no reliance can be placed on the presence of mere traces of sulphuric acid in clothing.

There would be great difficulty in administering sulphuric acid in a *solid* article of food; nevertheless such a case is recorded by Dr. Norman Chevers. (Med. Jur. for India, 1856, p. 165.) In May, 1853, a native of Pilibhut, while eating some sweetmeats which had been given to him, complained of a burning sensation at the back of his throat. He charged the man who gave them to him of having poisoned him. He died in four hours, having exhibited all the signs of irritant poisoning. On examining a portion of a sweetmeat, Dr. Monat found that it contained a considerable quantity of sulphuric acid. It is difficult to understand how the deceased could have taken sufficient to destroy life so rapidly under the circumstances. The process of analysis would be the same as for stains on clothing.

AROMATIC SULPHURIC ACID.

There is a preparation under this name in the Pharmacopœias of Edinburgh and Dublin; it is also known as elixir of vitriol, or acid elixir of vitriol. It is a mixture of sulphuric acid and rectified spirit (sulphovinic acid), to which ginger and cinnamon are added. According to the Dublin College, the sulphuric acid forms about one-tenth by measure. It is therefore a diluted form of sulphuric acid.

Symptoms.—One instance of poisoning by it has been reported by Mr. Blyth. A woman, æt. 25, swallowed *ten drachms* of the acid in mistake for a black draught. She experienced a sharp, burning, and disagreeable taste, great heat and pain in the stomach and gullet, accompanied with constant vomiting of a dark-colored liquid streaked with blood, with great difficulty of deglutition. Magnesia and water were freely given, so that before medical assistance had arrived, the acid was expelled from the stomach. In about eleven hours there was a considerable discharge of blood from the rectum. There was irritability of the stomach with thirst, and a copious discharge of saliva, but in two days the patient perfectly recovered. (Med. Gaz. xxv, 944.)

Analysis.—Owing to the presence of the spirit the specific gravity of this preparation is 0·974. The acid may be precipitated by the addition of carbonate of baryta: the precipitate, washed with nitric acid, and the residue calcined with charcoal, would indicate the presence of a sulphate by the production of a sulphuret. Alcohol might be separated by distillation, with or without the previous addition of carbonate of potash. The sulphuric acid forms rather more than one-tenth part by measure of this preparation.

QUANTITATIVE ANALYSIS.—It may be sometimes necessary to state how much sulphuric acid is present in a particular liquid. In order to determine this point, a portion of the liquid should be measured off, and the whole of the sulphuric acid present precipitated by the salt of baryta. The sulphate of baryta should be rendered pure by boiling it in nitric acid, then washed, dried, and weighed. For every one hundred grains of dried sulphate obtained, we must allow half the weight, *i. e.* fifty grains of common oil of vitriol (bihydrate) to have been present; hence the rule is a very simple one. As the equivalent of the bihydrated acid is 58, and that of sulphate of baryta 116, the proportion of acid is one-half of the weight of the precipitate. If we thus obtain the weight of the sulphuric acid present, it is very easy from its known specific gravity, to calculate the quantity by *measure*. The specific gravity of pure sulphuric acid is nearly twice that of water, being equal to 1·84; hence one ounce by measure will be equal to two ounces by weight. I have found that one fluid drachm, or one teaspoonful of a sample of common oil of vitriol (specific gravity 1·833) weighed 102·3 grains, and a tablespoonful 409·2 grains.

SULPHATE OF INDIGO. (*Sulphindigotic acid.*)

Several cases of accidental poisoning by this substance have occurred. As the compound is nothing more than a solution of one part of indigo in nine or ten parts of common oil of vitriol, the symptoms and appearances are the same as those which have been described for the latter substance. This kind of poisoning may be suspected, when, with these symptoms, the membrane of the mouth has a blue-black color. The vomited matters, as well as the fæces, are at first of a deep-blue tint; afterwards green; and it was observed in two instances that the urine had a blue tinge.

Symptoms.—One of the cases, reported by Orfila, was that of a child, which died in seven and a half hours. The other was observed by M. Bouchardat, and is of some interest. A young woman, aged 18, swallowed—as it was conjectured—about an ounce of the sulphate of indigo. Immediately afterwards she felt an acute burning pain in the throat and in the stomach. She threw herself on the ground, and her cries soon brought around her, her neighbors, who found her vomiting a bluish-colored liquid, which effervesced on the pavement. A quantity of oil and milk was immediately given to her; the milk was speedily thrown up coagulated, and of a blue color. When brought to the hospital, three hours afterwards, she was in the following condition: her face pale; features somewhat altered; her eyes were sunk, and her lips of a violet tinge. There was a yellowish-colored spot on the upper lip, at each angle of the mouth. The tongue was blue, the throat was painful, and there was a sense of constriction. The region of the stomach was tender. There was no pain in the abdomen; obstinate constipation; respiration difficult; great anxiety; coldness of the upper extremities; and a quick and small pulse. Her intellect was clear, and her answers to the questions put were sensible and proper. Four drachms of calcined magnesia were administered in a pint of water; much of this was rejected by vomiting accompanied by bluish clots. A few hours afterwards the pain in the throat was very severe,—the upper extremities were cold, and the pulse was imperceptible. The urine which she passed had a slight tinge of blue. She continued to become worse; the vomiting of chocolate-colored matter returned; and she died about ten o'clock—*i. e.* about eleven hours after having taken the poison.

Appearances.—The body was examined twenty-seven hours after death. The head presented no particular appearance. There was no sign of corrosion in the mouth. The mucous membrane of the throat and gullet was easily detached in dry, white, brittle, layers. The heart was filled with three ounces of coagulated blood; the aorta was also filled with brown and semi-liquid clots; the lining membrance of this vessel was of a bright red color! The stomach was distended, and contained two ounces of a brown-colored liquid. The mucous membrane was carbonized, and of the color of soot, with slight patches of redness throughout its whole extent, except for about an inch near the pylorus, where it was of a rose-red color. It was easily detached in layers, but there was no trace of ulceration. The membrane of the duodenum was inflamed and ulcerated, and in parts it was found corroded and blackened. A dark-colored mucus was seen in the small intestines, and patches of a blue color were scattered through the colon. The femoral arteries were filled with a semi-coagulated dark-colored blood. The cavity of the left femoral artery was completely obstructed by the clot. M. Bouchardat, who reports this case, considers that the deceased died from the absorption of the acid into the bloodvessels,—by which the blood was coagulated, and the circulation arrested. (See ante, p. 237.) Several instances of recovery are on record. Dr. Galtier reports two, —one of which is the case of a young woman who had swallowed rather more than an ounce of sulphate of indigo. She recovered on the eighth day. Calcined magnesia and milk were found to be the best remedies. (Toxicologie, i, 206.)

Analysis.—The process is the same as that described for sulphuric acid in organic mixtures (ante, p. 244). The blue color of the sulphate is immediately destroyed by boiling it with nitric acid. The barytic test may then be employed in the usual way.

CHAPTER XVI.

POISONING BY NITRIC ACID OR AQUA FORTIS—ACTION OF THE CONCENTRATED AND DILUTED ACID—NOXIOUS EFFECTS OF THE VAPOR—APPEARANCES AFTER DEATH—QUANTITY REQUIRED TO DESTROY LIFE—PERIOD AT WHICH DEATH TAKES PLACE—PROCESSES FOR DETECTING THE POISON IN PURE AND ORGANIC LIQUIDS—ON ARTICLES OF CLOTHING.

General Remarks.—THIS substance is popularly known under the name of *Aqua fortis*, or Red spirit of nitre. According to Tartra, it seems to have been first used as a poison about the middle of the fifteenth century. Although it is perhaps much more used in the arts than oil of vitriol, cases of poisoning by it are by no means so common. Tartra was only able to collect fifty-six cases, extending over a period of nearly four hundred years (Traité de l'Empoisonnement, 1802); and it appears from the return of inquisitions for 1837–8, there were only two fatal cases reported to have occurred in England during those two years. Cases of poisoning by this acid have been chiefly the result of accident or suicide. I have only met with one instance where it was poured down the throat of a child for the purpose of murder. The *external* application of nitric acid has been a criminal cause of death on several occasions: in one case, the acid was poured into the ear of a person while sleeping, and it led to the slow destruction of life. These are not strictly cases of poisoning, but more nearly approximate to death from wounding or mechanical violence.

SYMPTOMS.—*The Concentrated Acid.*—The symptoms, on the whole, are similar to those produced by sulphuric acid. They come on *immediately*, and the swallowing of the acid is accompanied by intense burning pain in the throat and gullet, extending downwards to the stomach: there are gaseous eructations, more copious than in poisoning by sulphuric acid, from the chemical action of the poison,—swelling of the abdomen, violent vomiting of liquid or solid matters, mixed with altered blood of a dark-brown color, and shreds of mucous and membranous flakes, of a yellow color, having a strong acid reaction, and a peculiar odor. The abdomen is generally tender; but in one well-marked case of poisoning by this acid, the pain was chiefly confined to the fauces: probably the poison had not reached the stomach. The mucous membrane of the mouth is commonly soft and white, after a time becoming yellow, or even brown; the teeth are also white, and the enamel is partially destroyed by the chemical action of the acid. There is great difficulty of speaking, as well as of swallowing, the mouth being filled with viscid mucus: the power of swallowing is sometimes entirely lost. Marks on the skin, from the spilling of the acid, are at first whitish, then yellow, and afterwards brown. On opening the mouth, the tongue may be found swollen and of a citron color; the tonsils are also swollen and enlarged. The difficulty of respiration is occasionally such, as to render tracheotomy necessary, especially in young subjects. (Case by Mr. Arnott, Med. Gaz. xii, 220.) As the symptoms progress, the pulse becomes small, frequent, and irregular; the surface of the body cold, and there are frequent rigors. The administration of remedies—even the swallowing of the smallest quantity of liquid, increases the severity of the pain, occasions vomiting, and gives rise to a feeling of laceration or corrosion. (Tartra, 144.) There is obstinate constipation, with occasionally suppression of urine. Death takes place in from eighteen to twenty-four hours, and is sometimes preceded by a kind of stupor, from which the patient is easily roused. The intellectual faculties, however, commonly remain clear until the last. In one instance the patient was insensible, but she ultimately recovered. Death may be occasioned by this acid, in consequence of its action on the larynx, as in the case of sulphuric acid. Should the patient survive the first effects of the poison, the mucous membrane of the throat and gullet may be discharged, either in irregular masses, or in the form of a complete cylinder of the œsophageal lining. (See ante, p. 227.) There is great irritability of the stomach, with frequent vomiting and destruction of the powers of digestion: the patient becomes slowly emaciated and dies from starvation or from exhaustion. In a case which proved fatal, in St. Bartholomew's Hospital, in March, 1851, the following symptoms were observed. A man swallowed about an ounce of nitric acid. He immediately vomited, but did not suffer much pain. When brought to the hospital, about half an hour afterwards, he looked pale and haggard, the skin was cold, and the pulse very feeble. The vomiting had quite ceased. He complained of no pain in the stomach, but of some uneasiness about the throat. He lay quiet for several hours, occasionally drinking a mixture of magnesia and water; he then became restless, and complained of severe pain in the abdomen, which increased in severity. He suffered greatly for three or four hours, and then died, having survived the taking of the poison about fifteen hours.

The following case occurred to Dr. Warren, U. S. A negress, æt. 34, wishing to destroy the child of which she supposed she was pregnant, took three drachms of nitric acid into her mouth, of which it is believed she swallowed two. Although alkalies and mucilaginous drinks were given to her, there was severe burning pain in the mouth with great restlessness and delirium. On the following day when examined at the hospital, yellow stains were observed upon her clothing, and the whole inside of the mouth and throat, so far as it could be seen, was of a deep yellow color, the tongue looking as if covered with Indian meal,—the respiration painful, labored, and stridulous, and speech almost impossible. The arms and legs were cold, the countenance of a leaden hue: the pulse 120 and very small. For the first four or five days she suffered from soreness of the mouth and throat, difficulty of swallowing, thirst, salivation, and some vomiting. She complained of tenderness of the abdomen, but not particularly over the stomach, walking with difficulty, and bent much forwards; but this may have depended on some violence which she had sustained on the day on which she took the acid. At the end of a week, she was recovering, and had some appetite. There had been no fever, but rather a state of depression. On the 12th day she was removed from the hospital. On the morning of the 14th day, she was attacked with cramps in the stomach, and excessive pain and tenderness, which were partially relieved by opiates. On the following morning, however, she was found dead, with a great quantity of blood in the bed about her, which she had apparently vomited. (Amer. Journ. Med. Soc. July, 1850, p. 36.) For the Appearances, see p. 256, post.

Vapor of the acid.—The *vapor* of this acid may destroy life. In March, 1854, Mr. Haywood, a chemist, of Sheffield, lost his life under the following circumstances: He was pouring a mixture of nitric and sulphuric acids from a carboy containing about sixty pounds, when, by some accident, the vessel was broken. For a few minutes he inhaled the fumes of the mixed acids, but it does not appear that any of the liquid fell over him. Three hours after the accident, he was sitting up and appeared to be in moderately good health. He was then seen by a medical man, and complained merely of some cuts about his hands. He coughed violently. In three hours more, there was difficulty in breathing with increase of the cough. There was a sense of tightness at the lower part of the throat, and the pulse was hard. At times, he said, he could scarcely breathe. He died eleven hours after the accident. On inspection, there was congestion of the trachea and bronchial tubes, with effusion of blood into the latter. The heart was flaccid, and contained but little blood; and the lining membrane of the heart and aorta was slightly inflamed. The blood gave a slightly acid reaction with test-paper. The larynx was not examined. It is very probable that the seat of mischief was in this organ, and that the deceased died from inflammatory effusion and swelling of the parts about the opening of the windpipe. (Lancet, April 15, 1854, p. 430.) The vapors produced by a mixture of strong nitric and sulphuric acids are of a most noxious and irritating kind. On one occasion, in preparing gun-cotton, I accidentally inhaled the vapor, and suffered from constriction of the throat, tightness in the chest, and cough for more than a week.

The diluted acid.—The symptoms above described apply to *acute* cases of poisoning by concentrated nitric acid. When the acid is *diluted*, they are somewhat modified according to the degree of dilution. A remarkably instructive case of poisoning by diluted nitric acid has been published by Dr. Puchelt, of Heidelberg; it shows not only the progress of the symptoms but also the powers of nature in resisting for a time the chemical destruction of an important organ. A man, aged 52, swallowed two ounces of diluted nitric acid (the strength not stated). He was *immediately* seized with severe burning pain in the mouth and throat; and this was followed by vomiting, whereby the greater part of the acid was probably ejected. He was not seen for several hours, and then the symptoms had so far subsided that the hospital assistant sent him away as not requiring immediate attendance. An oily emulsion was subsequently given to him. After the lapse of thirty-six hours, he was admitted into the hospital, and was for the first time seen by Dr. Puchelt. The mucous lining of the mouth and pharynx was covered with a white shreddy membrane, which could be readily peeled off; parts were already abraded. There were yellow stains on the cuticle around the mouth, especially upon the upper lip. The patient experienced great difficulty in swallowing; the breathing was laborious, the stomach tender, and the abdomen was hard and retracted. On the whole, the symptoms were very favorable, and led to the suspicion that but little injury had been done to the stomach. Leeches and other antiphlogistic means were employed, and in about eight days he began to retain a portion of food on the stomach. Nevertheless, his strength diminished, and he became emaciated; on the fifteenth day the food which he took was rejected; on the sixteenth, some blood was found mixed with the stools; on the seventeenth, there was great pain, with vomiting of black fluid blood, and of decomposed membrane of a fibrous structure, which, when spread out, was a foot in breadth. This membrane was marked with black spots, as if it were burnt, and perforated with numerous small and large apertures. A large quantity of black putrid blood was at the same time passed by stool. The symptoms became after this more unfavorable, and the vomiting of blood frequently recurred, until death took place on the *twenty-third* day after the poison had been swallowed. (See post, p. 257.)

There is no doubt that the diluted acid was in this case much stronger than that of the English Pharmacopœia, which contains about one-seventh by measure of strong nitric acid; or three parts of nitric acid to seventeen parts of water, its specific gravity being 1·082. I have not met with any instance of poisoning by this diluted acid.

APPEARANCES AFTER DEATH.—A full account of these will be found in the well-known work of Tartra, Essai sur l'Empoisonnement par l'Acide Nitrique, published An x.

Supposing death to have taken place rapidly, the following appearances will be met with. The skin of the mouth and lips will present various shades of color, from an orange-yellow to a brown; it appears like the skin after a blister or burn, and is easily detached from the subjacent parts. Yellow spots produced by the spilling of the acid, may be found about the hands and neck. A yellow frothy liquid escapes from the nose and mouth, and the abdomen is often much distended. The membrane lining the mouth is sometimes white, at others of a citron color; the teeth are white, but present a yellowish color at their junction with the gums. The pharynx and larynx are much inflamed; the latter sometimes swollen. The lining membrane of the gullet is softened, and of a yellow or brown color, injected (contains more blood), is easily detached, often in long folds. The windpipe is more vascular than usual, and the lungs are congested. The most strongly-marked changes are, however, seen in the stomach. When not perforated, this organ may be found distended with gas,—its mucous membrane partially inflamed with patches of a yellow, brown, or green color, or it may be even black. This green color is due to the action of the acid on the coloring matter of the bile; but it must be remembered that a morbid state of the bile itself often gives this appearance to the mucous membrane in many cases of death from natural disease. There is occasionally inflammation of the peritoneum, and the stomach is glued to the surrounding organs. Its coats are often so much softened, as to break down under the slightest pressure. In the duodenum similar changes are found; but in some cases the small intestines have presented no other appearance than that of slight redness. It might be supposed that the stomach would be in general perforated by this very corrosive substance; but this is far from being the case. Tartra only met with two instances, and in one of these, the person survived twenty, and in the other thirty hours. In giving this poison to rabbits, I have not found the stomach perforated, although the acid had evidently reached that organ, from its coats being stained of a deep-yellow color. In these experiments the non-perforation appeared to be due to the protective influence of the food with which the stomach was distended. In the few cases that are reported in English Journals, the stomach has not been perforated; the poison was swallowed soon after a meal, and its coats had thus escaped the corrosive action of the acid. In the case which proved fatal at Bartholomew's Hospital (ante, p. 253), the stomach was extensively destroyed: the surface was not stained yellow, but the mucous membrane was removed by corrosion, and the coats beneath were partly reddened and partly blackened, as a result of the action of the acid on the blood in the vessels.

In Dr. Warren's case (ante, p. 253), there was a large smooth yellow-colored patch on the tongue, and there was redness on the epiglottis. For the first two inches the gullet was healthy; but below this it was found very soft, of a greenish color internally, and purple externally, and full of coagulated blood. The stomach was purple externally, and adherent to the neighboring parts by recent lymph, except at the left extremity, where there were old and close adhesions to the spleen: internally it was of a greenish-yellow color, emphysematous (having a blistered appearance), and so much softened that it could not be separated from the surrounding parts without giving way in every direction. The fore part of the stomach was detached from the rest of the organ to a great extent, when the coverings of the abdomen were raised. The stomach was filled with recently coagulated blood, and the open orifices of several vessels were distinctly seen on the inner surface. The intestines contained blood throughout the first two or three feet, but they were otherwise uninjured (Am. Jour. Med. Sci. July, 1850, p. 36). In a case which occurred at the Hôtel Dieu at Lyons, the stomach was distended with gas and perforated at the greater end, the opening being partially plugged by the spleen, which had become adherent over it. In the small intestines there were numerous sloughs. In Mr. Arnott's case, a boy, æt. 13, supposing that he was going to drink beer, swallowed a mouthful of a fluid which proved to be nitric acid. Acute pain was felt in the mouth and throat. Magnesia was administered, and vomiting was quickly induced. The vomited matters consisted of a large quantity of food partly digested. There was great constitutional depression, but the chief distress was from symptoms indicative of inflammation of the larynx. Mr. Arnott performed the operation of opening the larynx with some relief to the boy, but he died in thirty-six hours from the time of swallowing the acid. On inspection sixteen hours after death, the effects of the acid were found to be confined to the tongue, palate, fauces, tonsils, and lining membrane of the throat and gullet. None of the acid had entered the larynx, but there was a layer of coagulated lymph on the mucous surface of the windpipe, arising from inflammation which had extended from the parts adjacent. The base, edges, and tip of the tongue, with the lower part of the gullet, were deprived of their investing membrane. The portion of membrane which remained adherent had a citron color. That which covered the tongue was ragged at its edges, that of the throat and gullet was dry, corrugated, and marked with longitudinal and transverse lines. It could everywhere be readily stripped off, the part beneath appearing red. The edges of the glottis were swollen, the epiglottis was destroyed. There was no trace of the effects of the acid in the stomach except at the lesser end, where the orifices of the mucous glands presented a citron color like that of the throat. The mucous membrane of the stomach was probably protected from the action of the acid by the quantity of food contained in the organ (Roupell on the Effects of Poisons, pl. iv; Med. Gaz. vol. xii, p. 220, and vol. xiv, p. 489).

In cases of *chronic* poisoning, *i. e.* where death takes place *slowly*, the appearances are of course different, as the following case will show. A man, aged 34, swallowed a wine-glassful of nitric acid, but the greater portion was immediately rejected by vomiting. An attack of acute gastritis followed, which was combated by the usual remedies. The man was discharged from the hospital into which he had been admitted, in three weeks; but about a month afterwards, he was readmitted, in consequence of his suffering severe pain extending down the gullet to the stomach, as well as from vomiting after taking food. The patient gradually sank, and died *three months* after he had taken the acid. On dissection, the pylorus was found so diminished in size, that its diameter did not exceed a line or two, and the duodenum was equally contracted for about an inch and a half from its commencement. The mucous membrane was softened and red in patches; and there were several cicatrices of ulcers. The subjacent tissues were in a scirrhous state. (See Med. Chir. Rev. vol. xxviii, 553.) As a contrast to this, the appearances met with in a case of poisoning by a similar dose of nitric acid which proved *rapidly* fatal may be here described. A man drank about two ounces of aqua fortis, and died speedily from its effects. On examining the body, the lips were found partly yellow, and partly of a brownish-red color, dried up like parchment. Several yellow or parchment-colored spots were observed on the chin, as also on the cravat. The mucous membrane of the mouth was white and easily detached,—that of the tongue was dry and hard,—that of the pharynx and œsophagus yellowish-green in color and of a leathery consistency. The stomach contained a dark-colored liquid, highly acid. It was externally mottled of a greenish-blue and black color. The mucous membrane throughout was softened, and in a gangrenous state. The same appearances were met with, although in a less degree, in the duodenum and upper part of the ileum. On analysis, the contents of the stomach yielded nitric acid. (Von Raimann, Medicinisch. Jahrb. 20 B. 2 S. p. 221.)

In Dr. Puchelt's case (ante, p. 254), in which death took place on the twenty-third day, when the abdomen was opened there was no appearance of a stomach, but in its place a cavity formed by the liver, colon, and other viscera; the interior wall, lesser curvature, and upper part of the posterior wall, being wholly absent. A dark green mass was spread over the interior; but the parietes were so soft as to give way on the slightest pressure. The intestinal canal, with the exception that it contained a large quantity of bloody matter, presented nothing peculiar. The mucous membrane of the gullet was found removed throughout its whole length. (Ein Fall von Vergiftung mit Scheidewasser, von Dr. F. A. B. Puchelt, Heidelberg, 1845.)

FATAL DOSE.—The remarks made on this subject in speaking of sulphuric acid (ante, p. 238), apply here. Tartra states that the quantity usually taken varies from one or two drachms to four ounces; but in most of the cases which he reports, the quantity taken is not mentioned. Indeed, the obtaining of any information of this kind is purely accidental; and the determination of the exact quantity swallowed, must be therefore very difficult. One point is certain; a similar dose will not kill two persons in the same time,—one may die slowly, and the other rapidly (supra), according to whether the stomach at the time contains food or not. The *smallest* quantity which I find reported to have destroyed life, is about *two drachms*. It was in the case of a boy, aged 13; he died in about thirty-six hours. In Dr. Warren's case, a woman died from a similar dose in fourteen days (ante, p. 253). But less than this,—even one drachm, would doubtless suffice to kill a child; and, under certain circumstances, an adult; for the fatal result depends on the extent of the mischief produced by this corrosive poison in the throat, windpipe, and stomach. What is the largest dose of concentrated acid from the effects of which a person has recovered, it is difficult to say; since in most cases of recovery mentioned by authors, the quantity of the poison actually swallowed was unknown.

In one instance, a woman, æt. 26, recovered in a few days after having swallowed *half an ounce* of aqua fortis of the usual strength. There was great reason to believe, either that the poison did not reach the stomach, or that it produced but little action on the organ. The chief seat of pain was in the throat and gullet. (Lancet, May 8, 1847, p. 489.)

PERIOD AT WHICH DEATH TAKES PLACE.—This must depend on the quantity swallowed, the strength of the acid, and whether any medical treatment has or has not been adopted. Out of twenty-seven deaths from nitric acid, reported by Tartra, in nineteen it destroyed life rapidly, and in eight slowly. This author met with two instances in which death took place within *six* hours after the poison was swallowed; but he considers that the greater number who fall victims to the direct effects of the acid, die within twenty-four hours. Sobernheim relates a case of poisoning by nitric acid, which proved fatal in *one hour and three-quarters*. (Op. cit. 402.) This I believe to be the most rapidly fatal case on record, where the acid acted as a poison. The usual well-marked effects were found in the gullet, stomach, and duodenum. In infants, however, life may be destroyed by this poison in a few minutes, should it happen to affect the air-passages. A woman, shortly after her delivery, in the absence of her attendants, poured a quantity of nitric acid into the mouth of her infant. The mother concealed from those about her this attempt at murder; but medical assistance was immediately sent for. The child died in a *few minutes*. Some of the acid had been spilled; and from the yellow color of the stains, the medical man suspected that the child had been poisoned by aqua fortis. On inspection, nitric acid was found in its stomach, and the mother confessed the crime. (Cazauvieilh, Du Suicide et de l'Aliénation Mentale, p. 274.) Although in the report of this case the condition of the throat and larynx is not stated, it is highly probable, from the rapidity with which death took place, that this event was in great part due to suffocation. The following experiment will perhaps serve to show how speedily life may be destroyed under these circumstances. Half a drachm of concentrated nitric acid was given to a rabbit. In about half a minute, it became insensible, and apparently lifeless. There was no sign of pain or irritation. It died in *one minute.* A small quantity of gaseous matter, in the form of a fuming vapor, escaped from the nostrils. The abdomen was much swollen before death. On inspection, it was found that the poison had strongly acted on and corroded the parts about the larynx. A portion had penetrated into the lungs, turning them yellow, and corroding them. Another portion had been swallowed, and had entered the stomach, producing the usual yellow stains, but it had not perforated the organ. With regard to the *longest* period at which death has taken place from the effects of this poison, a case has been already related, where a man who had swallowed nearly two ounces, did not die until three months afterwards (ante, p. 257). The longest case is perhaps that recorded by Tartra, in which a woman died from exhaustion, produced by the secondary effects of the poison, *eight months* after having swallowed it.

TREATMENT.—It may be the same as that recommended in poisoning by sulphuric acid. In addition to the remedies there suggested, a diluted solution of carbonate of soda with barley-water, and other demulcents, may be administered. In many cases, there is an utter impossibility of swallowing even the smallest quantities of liquid; and if an attempt be made to introduce these remedies by a tube, there is great risk of perforating the softened parietes of the pharynx, larynx, or gullet. Should suffocation be threatened, then tracheotomy may be resorted to. Modern experience is rather adverse to the recovery of these cases under any form of treatment; but according to Tartra, in accidental poisoning by this acid, there is great hope of recovery, if the patient receive timely assistance. He states that out of thirty-one cases, twenty-three recovered,—seventeen perfectly; while out of twenty-four cases, wherein suicide was attempted, only six recovered. (Op. cit. p. 186.)

CHEMICAL ANALYSIS.

Nitric acid may be met with either concentrated or diluted. The *concentrated acid* varies in color from a deep orange red to a light straw yellow. The sp. gr. of a sample of the acid was 1·392. A teaspoonful was equivalent to 79 grains, and a tablespoonful (half an ounce) to 316 grains. It may be recognized,—1. By evolving acid fumes when exposed. 2. By its staining organic matter yellow or brown, the color being heightened and turned to a reddish tint by contact with caustic alkalies. 3. When mixed in the cold with a few copper cuttings, it is rapidly decomposed—a deep red acid vapor is given off, and a greenish-colored solution of nitrate of copper is formed. Tin or mercury may be substituted for copper in this experiment. 4. It does not dissolve gold leaf even on boiling; but on adding a few drops of hydrochloric acid the gold is immediately dissolved.

In the *diluted* state. This acid is not precipitated like the sulphuric by any common reagent, since all its alkaline combinations are soluble in water. 1. The diluted liquid has a highly acid reaction, and on boiling it with some copper turnings, red fumes of nitrous acid vapor are given off, unless the proportion of water is too great. At the same time, the liquid acquires a blue color. 2. A streak made on white paper with the diluted acid, does not carbonize it when heated; but a scarcely visible yellow stain is left. Diluted sulphuric and hydrochloric acids carbonize paper under similar circumstances. 3. The liquid is neither precipitated by nitrate of baryta nor by nitrate of silver. These two last experiments give merely negative results—they serve to show that the sulphuric and hydrochloric acids are absent. A portion of the acid liquid should now be evaporated, in order to ascertain whether it is entirely free from saline matter—*e. g.* a nitrate. If it is simply an acid liquid, and contains no nitrate, it may be carefully neutralized with potash, and then evaporated slowly to obtain crystals. If the liquid contained nitric acid, these crystals will possess the following characters: 1. They appear in the form of lengthened fluted prisms, which neither effloresce nor deliquesce on exposure. One drop of the solution evaporated spontaneously on glass will suffice to yield distinct and well-formed crystals. This character distinguishes the *nitrate* of potash from a large number of salts. When neutralized with soda, the crystal is of a rhombic form,—a very striking (microscopic) character of *nitrate* of soda. 2. When moistened with strong sulphuric acid, the powdered crystals slowly evolve a *colorless* acid vapor. By this test, the nitrate is known from every other deflagrating salt. 3. A portion of the powdered crystals should be placed in a small tube and mixed with their bulk of fine *copper* filings. The mass is then to be moistened with water, and a few drops of strong *sulphuric acid* added. Either with or without the application of a gentle heat, a decomposition ensues, by which red fumes of *nitrous acid* are evolved, recognizable by their color, odor, and acid reaction. If a tube only one-eighth of an inch in the bore be used for this experiment, one-tenth of a grain of nitrate of potash will give satisfactory results. This is equivalent to about one-twentieth of a grain of nitric acid—a quantity to which the toxicologist will not often have to confine his analysis in medico-legal practice. Should the quantity of suspected nitrate be very small, it may be placed in a dry Florence flask with a few cuttings of copper, and a few drops of strong sulphuric acid poured in the mixture. A piece of paper soaked in a mixture of starch and iodide of potassium may then be suspended by a closely fitting cork in the neck of the flask. Sooner or later, and without the aid of heat, nitrous acid will be evolved, and, although the color of the fumes will not be apparent, the production of blue iodide of starch in the paper will indicate their presence, and prove that the salt was a *nitrate*. This mode of testing by copper and sulphuric acid is open to objection if any alkaline chloride be mixed with the suspected nitrate. When this mixture exists (a fact demonstrable by the use of nitrate of silver to a solution of the salt), we may then resort to 4. Add a piece of gold leaf, and a few drops of strong and pure hydrochloric acid to the suspected salt in a tube, and warm the mixture by a spirit lamp. If a nitrate be present, the gold is dissolved wholly or in part; and in order to prove that this solution has taken place, a few drops of chloride of tin may be added to the mixture. If any gold is dissolved, the liquid will acquire a pink or dark purple-brown color; otherwise there will be no change of color. The presence of an alkaline chloride does not interfere with this result, but rather aids in the solution of the gold. It must be remembered that a chlorate, bromate, or iodate, will dissolve gold under similar circumstances; but the addition of sulphuric acid to these liberates a colored gas or vapor and a peculiar odor. (See 2.) The analyst must be careful to use hydrochloric acid free from any trace of nitric acid. It should be tested with gold leaf previously to adding it to the suspected salt. By the use of either copper or gold, nitric acid or a nitrate, even in minute quantity, may be readily detected.

Objections.—There are no practical objections which can be urged to the mode of testing for nitric acid above recommended. When the copper and gold tests give the results described, the presence of nitric acid or of a nitrate may be considered as proved.

In liquids containing organic matter.—Nitric acid precipitates and combines with albumen and casein. It may be presented for analysis in such liquids as vinegar, tea, or porter. In this case, besides the acid reaction, there will be the peculiar smell produced by this acid when mixed with substances of an organic nature. The usual test may here fail, for unless the quantity of nitric acid in such a liquid as porter be considerable, the orange-red fumes of nitrous acid are not evolved on boiling it with copper cuttings. Care must be taken in examining such liquids, that we do not fall into the error of pronouncing nitric acid to be present, when any acid article of food is simply mixed with a nitrate. A little nitre mixed with vinegar or porter—both of which liquids have an acid reaction—might deceive an analyst, unless he adopted the precaution recommended under sulphuric acid (ante, p. 244), *i. e.* of evaporating to dryness a portion of the suspected liquid, and examining for the presence of a nitrate the dry residue, which may be obtained. If no dry saline residue is procured, we may safely proceed to obtain evidence of the presence of nitric acid by the process mentioned below. If the liquid be viscid, this viscidity must be destroyed by dilution with water: and, in all cases, if any solid or insoluble substances are floating in it, as in the *matters vomited* or *contents of the stomach*, it must be filtered, in order to obtain at least a portion of the acid liquid. This operation is slow, especially if the liquid has been boiled. If we procure a clear acid liquid, the color may be disregarded. We should then carefully neutralize it with a weak solution of bicarbonate of potash, and concentrate to a small bulk by evaporation. As a trial test we may dip in a slip of bibulous paper, dry it, and observe whether it burns with deflagration. This commonly answers, unless the quantity of nitric acid present be very small, or unless the nitrate of potash formed, be mixed with a large portion of some other salt. The liquid may now be set aside for the deposit of crystals, which are deposited more or less colored and impure. The presence of this impurity does not at all interfere with the action of the most important test for nitric acid, namely, the mixture of copper and sulphuric acid either in a tube or in a Florence flask, as already described (ante, p. 260). The crystals may, however, if necessary, be purified by digesting them in ether or alcohol. These liquids do not dissolve the nitrate of potash, but they may serve to remove from it the organic matters by which it is colored. The draining of the crystals on blotting paper, and a second crystallization from water, will render them sufficiently pure for the determination of the presence of nitric acid. For the application of the gold test, it is desirable to have the crystals in as pure a state as they can be obtained.

Neutral liquids.—But the vomited matters and the contents of the stomach may have no acid reaction, and yet nitric acid be present. Thus it may have become neutralized by lime or magnesia, through the administration of antidotes. In such a case, it would not be easily detected, if present only in small quantity. Nitrate of lime may be dissolved out by alcohol, or by the addition of carbonate of potash to the filtered neutral liquid previously concentrated by evaporation, either of these earthy nitrates may be transformed to nitre, and the analysis then proceeded with,—the carbonate of lime and magnesia formed being separated by filtration. Again, for the same reasons as those mentioned in speaking of sulphuric acid, the liquid found in the stomach of a person who has died from nitric acid, may not contain a trace of the poison, either free or combined. Its absence, therefore, does not negative a charge of poisoning. Nitric acid has a much stronger tendency than the sulphuric, to combine with the solid organic tissues; and in decomposing them, it undergoes decomposition itself. In a case of this kind, those parts of the mucous membrane, whether of the gullet or stomach, which are stained yellow or corroded, should be digested in water for some hours, at a gentle heat. If the acid is in well-marked quantity, cold water will suffice. The resulting liquid should be filtered and neutralized by potash, and subsequently examined for nitre. This experiment, from the small quantity of free acid present, is very likely to fail. In examining the stomachs of rabbits killed by nitric acid, I have found that even deeply stained portions of the mucous membrane have yielded commonly only faint traces of acid. But the discovery of no more than *traces* of acid in these cases of poisoning, is, in my opinion, tantamount to a failure of the chemical branch of evidence; for no inference could be drawn from such minute results relative to the fact of poisoning, unless the evidence from symptoms and appearances, with moral circumstances, were sufficiently conclusive; and when this is the case, whether the poison be wholly absent, or exist only in "infinitesimal" traces, it is a matter of trifling importance. The nitrates, although frequently used medicinally, are not natural constituents of the secretions of the alimentary canal.

Absorption and Elimination.—Orfila states that he has separated nitric acid from the urine of animals poisoned by diluted nitric acid.

He distilled the urine with sulphuric acid, neutralized by potash the acid liquid thus obtained, and succeeded in procuring nitrate of potash. The result was not uniformly the same. At certain stages of the poisoning only, the urine was found to contain nitric acid (Toxicologie, 1852, i, 185). As the nitrates are not constituents of the urine, the fact may be of importance, although the circumstances under which nitric acid was thus procured, are not likely to present themselves in a case of acute poisoning in a human being.

In a dead body which has undergone putrefaction, nitric acid may exist only as nitrate of ammonia—a salt, which is diffused through the soil of a burial-ground, and may, by percolation of water, be carried into and impregnate a dead body. The presence of traces of nitrates, under such circumstances, will amount to nothing, unless the appearances produced by nitric acid are found in the body. It will be proper in all cases of exhumation to examine the soil for nitrates.

On solid organic substances. Stains on clothing.—This acid is sometimes maliciously thrown at persons; and we may be required to examine some article of dress suspected to have been stained by it. The spots produced by strong nitric acid on woollen stuffs, are either of a yellow, orange-red, or a brown color, according to the time at which they are seen. On black cloth they speedily acquire a light yellowish-brown color, passing after a few days to a dingy olive-green with a red border. After a time they become dry (unlike those produced by strong sulphuric acid), and the texture of the cloth is entirely destroyed. In order to examine them, the stained portions may be cut out and digested in a small quantity of warm distilled water or in lime-water. If nitric acid is present, distilled water will acquire an acid reaction; but, in order to prove this, the liquid must be neutralized by bicarbonate of potash or lime, then evaporated to dryness, and the dry saline residue, if any, examined by the copper and gold tests for nitrate of potash or lime. Should the water acquire no acid reaction, then there is no perceptible quantity of nitric acid present. To render this certain, however, the water may be made feebly alkaline by potash, and again boiled with the stuff; the liquid may be filtered and examined for nitre. It is rare that any evidence of the presence of nitric acid is obtained by the latter process, when the stained portions of cloth do not give out any free acid to the distilled water in the first instance. Should any traces of nitric acid be perceived in an experiment of this kind, an unstained portion of cloth or stuff must be examined, before we can draw the inference that nitric acid has been spilled or thrown on it. On these occasions we may be often disappointed in searching for chemical evidence of nitric acid. Not to mention, that the acid may be easily removed by washing while the discoloration remains, we must remember that the acid is volatile, easily decomposed, and its nature entirely changed by contact with the organic substance. These facts will explain to us, why after a few weeks the chemical evidence of the presence of this acid is sometimes entirely lost; while, in the case of sulphuric acid, the stains may furnish abundant evidence of its presence after many years' exposure. In all cases of the suspected throwing of nitric acid, the spots on the dress should be examined as soon as possible, or a chemical analysis will be of no avail. The following case occurred at Guy's Hospital: A man had some strong nitric acid maliciously thrown in his face, and the sight of one eye was thereby entirely destroyed. He wore at the time a blue stuff coat, which was not sent to be examined, uutil *five weeks* after the accident, and only a few days before the trial of the prisoner for the offence! The sleeve and body of the coat were found to be covered with numerous spots of a yellowish-brown color. The spots were quite dry; they had evidently been caused by some corrosive acid. The color was discharged, and the fibre of the stuff corroded. Not a trace of nitric acid could be detected in them, although there was no reasonable doubt that it had been used. Its disappearance was probably due partly to its decomposition in the stuff, and partly to its volatility. Had the coat been examined soon after the offence, the nature of the acid would have been easily determined. I have been able to procure certain evidence of the presence of nitric acid in stains on black cloth, a fortnight after the liquid had been spilled. The quantity of acid present was, however, small. Dr. Christison has obtained evidence of the presence of this acid in stains on cloth, made seven weeks before (op. cit. 178); and Orfila states that he has found stains on felt, cloth, leather, and human skin, to retain an acid reaction for twelve or fifteen days. He detected nitric acid in the stains, by allowing the material to soak for some hours in a cold weak solution of bicarbonate of soda. The dry saline residue obtained, on evaporating the liquid, contained a nitrate. (Orfila, Toxicol. i, 187.) In conducting an analysis of this kind, it has been recommended, when we obtain an acid liquid, to test it with the nitrate of baryta and nitrate of silver. The liquid, if it contain nitric acid only, should give negative results; but there are few specimens of cloth which do not yield traces of sulphuric and hydrochloric acids, or of sulphates and chlorides, so that nitric acid may still be present when one or the other of these tests is affected. The spots produced by this acid on the human skin are at first yellow, and become of a deeper orange-yellow when touched with an alkali. By this they are known from spots produced by iodine, which are immediately bleached and removed by an alkali.

Quantitative analysis.—Convert the nitric acid contained in a measured quantity of the liquid, to nitre, by the process above described. Convert the whole quantity of nitre thus obtained to sulphate of potash, by the cautious addition of sulphuric acid. Dissolve this salt in water, and evaporate to dryness. Calcine the residue, and afterwards wash it with alcohol, to remove if necessary any portion of free sulphuric acid. For every one hundred grains of dry sulphate of potash thus obtained, we may estimate that there were present in the measured quantity of liquid, about eighty-two grains of liquid nitric acid or aqua fortis (bihydrate)—the bulk of which, by measure, may be easily determined by calculation. Should the dried sulphate be acid, it may be necessary to moisten it with a solution of sesquicarbonate of ammonia, and re-expose it to heat, to drive off the volatile alkali with the surplus sulphuric acid. Sulphate of potash, it must be remembered, is a perfectly neutral salt.

CHAPTER XVII.

POISONING BY MURIATIC ACID OR SPIRIT OF SALT—RARELY TAKEN AS A POISON—SYMPTOMS—APPEARANCES AFTER DEATH—FATAL DOSE—CHEMICAL ANALYSIS—DETECTION OF THE ACID IN PURE AND MIXED LIQUIDS—ON ARTICLES OF CLOTHING—IN CASES OF FORGERY—PHOSPHORIC ACID—EXPERIMENTS ON ANIMALS—ACTION OF PHOSPHOROUS ACID—TESTS FOR PHOSPHORIC ACID.

BUT little is known concerning the action of muriatic or hydrochloric acid as a poison. In the Coroners' return for England, during the years 1837–8, out of five hundred and twenty-seven cases of poisoning, there was not one in which this acid was the poison used. Only *three* cases of poisoning by muriatic acid occurred in this metropolis, during a period of sixteen years. From this statement it will be seen that the medico-legal history of this kind of poisoning is necessarily incomplete.

SYMPTOMS.—From the few reports that have yet appeared, the symptoms produced by this acid do not differ widely from those caused by the two other mineral acids, described in the preceding chapters. There is the same sensation of burning heat, extending from the throat to the region of the stomach, with vomiting of a highly acid liquid of a dark color, mixed with mucus and altered blood. The tongue is swollen and dry; and with much thirst, there is great difficulty of deglutition. The tonsils and throat are inflamed. An escape of acrid pungent vapors from the mouth, when the acid has been swallowed, is described by Orfila among the earliest symptoms; after an hour or two this has not been observed. In two cases, neither the vomiting nor pain in the abdomen was urgent, although both terminated fatally. The chief seat of pain was in the throat. In one instance, in which probably an ounce of the acid had been swallowed, the person was able to walk to his home at a distance of three-quarters of a mile. The pulse has been found small, frequent, and irregular; the skin cold and clammy. The intellectual faculties have remained clear until death. In the case of a Hindoo, Sinivassin, æt. 28, reported by Dr. Collas, the symptoms, about twelve hours after two ounces of the acid had been swallowed, were as follows: the head was drawn backwards, the mouth half open, the lips and face presented no spot or stain, the gums were pale, the teeth not discolored, the tongue was deprived of a strip of its investing membrane about the centre. The skin was cold, the pulse small and frequent, the breathing difficult, the abdomen painful. There was suppression of urine, but no purging. Magnesia with soap and water had been given to him, and were retained on the stomach. It was ascertained that the poison had been taken by mistake for brandy, and that there had been violent vomiting, the vomited matters effervescing on the floor. (Ann. d'Hygiène, Janvier, 1858, p. 209.) This case proved fatal. Mr. Procter, of York, communicated to me the particulars of a case in which a female, æt. 29, swallowed half an ounce of commercial hydrochloric acid. She was seen an hour and a half afterwards. She then complained of intense burning pain in the throat and along the gullet, but there was only slight pain in the stomach; and but very little tenderness of the abdomen. There was incessant vomiting. Magnesia and barley-water were freely given; but in half an hour there was collapse, rendering the use of stimulants necessary. In the evening, reaction was established; but the voice could scarcely be heard, and there was great pain in the throat. This was relieved by a few leeches, and the woman recovered in a fortnight. In this instance, the action of the poison appears to have been chiefly spent on the throat and gullet. (Guy's Hosp. Reports, 1851, p. 211.) Another case, reported by Dr. Allen, presents a more complete history of the symptoms from a larger dose. A girl, æt. 20, swallowed an ounce of hydrochloric acid on an empty stomach, with the intent to destroy herself. Vomiting had occurred, and alkaline remedies were prescribed before she was seen by Dr. Allen,—two hours after the poison had been taken. The countenance was pale and anxious: there was pain with burning heat in the throat and abdomen; the region of the stomach was very tender on pressure, the skin was cold, the pulse 130, small and thready, the tongue pale and whitish, and the throat much inflamed. She vomited freely a fluid of a brownish color, which was quite neutral. Barley-water and carbonate of soda were given. In six hours from the time of taking the poison, she vomited about half a pint of a bloody fluid. Vomiting of blood continued for about twelve hours. On the following day, there was great tenderness in the region of the stomach, with inflammation in the throat and pain in swallowing. In three days there were cramps and twitchings of the limbs, and a sense of coldness in the legs, although these felt quite warm. She then gradually improved: on the 15th day the pulse was 80; and she could swallow fluids without difficulty. There was still great tenderness over the stomach. (Medical Gazette, 1849, vol. xliv, p. 1098.)

In the follwing case the patient, a female, æt. 24, did not die for a period of eight weeks after taking more than two ounces of this acid (1000 grains). The immediate symptoms were: severe pain with a sense of burning in the tongue, back of the mouth, and gullet, as far as the stomach; a feeling of suffocation, escape of white vapors, and vomiting of a liquid which effervesced as it fell on the pavement. In three hours she was brought to the Hôtel Dieu; and it was found that the vomited matters had a brown and bloody appearance. Vomiting continued throughout the night to the extent of four quarts of a reddish liquid with solid masses of a red-brown color. These vomited matters had no acid reaction; on the next morning, the tongue and throat were covered with a white pellicle, and in parts the membrane was removed, as if by the corrosive action of the acid. The inner surface of the cheeks, the roof of the mouth, and under part of the tongue, presented no change. There was a severe burning pain in the throat, extending to the stomach, increased by pressure; but the acid did not appear to have reached the intestines. There was a copious discharge of saliva with shreddy masses of mucus, and any attempt to swallow was followed by spasms in the throat. The voice was feeble and hoarse, breathing quiet, pulse 96, regular and full, skin warm and dry, urine scanty; no evacuation from the bowels. On the second day there was delirium followed by paralysis of the limbs and collapse. During the eight weeks that the patient survived, there were variable symptoms, chiefly referable to the throat, lungs, and stomach (Annales d'Hygiène, 1852, vol. ii, p. 415, case by Dr. Guérard).

APPEARANCES AFTER DEATH.—The throat, larynx, and gullet, have been found highly inflamed, the mucous membrane lying in detached masses or actually sloughing away. In one instance the membrane was thickened. The coats of the stomach have been so much corroded that, in many places, there was only the peritoneal coat left; and in attempting to remove the organ in this case, the parietes gave way. The contents have been sometimes of a yellowish, at others of a dark-green color. In a case, in which the fundus of the gall-bladder had come in contact with the stomach, it was observed to have a bright-green color, arising from the well-known action of this acid on the bile. On removing the contents, the lining membrane has been found blackened, and presenting a charred appearance: the blackening extended through the whole length of the duodenum, and was especially marked on the prominent parts of the numerous valvulæ conniventes (folds of mucous membrane), the intervals being stained of a greenish-yellow color, from the action of the acid on the bile. (Case by Mr. Quekett, Med. Gaz. xxv, 285.) When death did not take place until after the lapse of several days, the coats of the stomach were of a dark color, highly inflamed, and for the most part in a sloughing state; large dark shreds of membrane were hanging from the sides of the organ, especially about the pylorus. The inflammation had extended also into the duodenum.

Perforation of the stomach has not been a common appearance. The mucous membrane of this organ has been found more or less corroded, and sometimes entirely destroyed. In a case referred to by Dr. Galtier (Toxicologie, vol. ii, p. 217, 1855), which was the subject of a criminal trial in 1846, the stomach was entirely disorganized, softened, and presented posteriorly several perforations, of different diameters, with rounded, thickened, and inflamed margins, adhering to the adjoining viscera by slight albuminous deposits. The pyloric orifice was thickened, as well as the mucous membrane of the small intestines. The large intestines were healthy. The mucous membrane of the throat was thickened, injected, and, on pressure, purulent matter escaped from it. The gullet was thickened throughout its extent, and its mucous membrane was in a state of suppuration. These appearances may be taken as representing the effects produced by the acid when the case is protracted. The patient in this case died eight weeks after she had taken the acid. The quantity taken was unknown. (See also Orfila, Toxicologie, vol. i, p. 216.)

In Dr. Collas's case (ante, p. 265), death took place in about twenty-four hours, and the inspection was made thirteen hours afterwards. Although the temperature was high, there was no odor, and no sign of putrefaction. The mouth and throat presented no alteration. The mucous membrane of the tongue was reduced to a grayish pulp, and was easily removed. The membrane of the gullet was rough and disposed in longitudinal folds. The upper and lower portions of the tube were dark-colored, but not carbonized; while the middle portion was pale. The stomach was distended, and presented externally red, green, and black discolorations. It contained about seven ounces of a black turbid liquid. In nearly its whole extent, the surface of the stomach was blackened, and the mucous membrane detached; in the vicinity of the pylorus, it was of a dull wine-red color. The duodenum was healthy, contrasting strongly with the condition of the stomach; this, as well as the other small intestines, contained a yellowish-colored liquid. The cavities of the heart, and the large arteries, contained red firm clots of blood, moulded to the form. The urine was acid, and yielded, by precipitation, a quantity of chloride, indicative, as it was supposed, of the presence of hydrochloric acid—about 0·9 per cent. (Ann. d'Hygiène, Janvier, 1858, p. 209.)

In Dr. Guérard's protracted case (ante, p. 266), in which death took place after eight weeks, the mucous membrane of the gullet was found swollen and softened throughout. At the upper part, the lining membrane was entirely removed; at the lower, it had a slate color. The mucous membrane of the stomach was softened and gelatinized with a brownish discoloration at the greater end; the muscular coat was laid bare in several places. The pylorus (intestinal opening) was hardened, contracted, and of a brown color. The peritoneum was covered with some false membranes. The small intestines were slightly injected. The whole of the parts about the larynx, epiglottis, and trachea, were much injected, and of a brown color. A quantity of serum was found in the left pleura, and the lungs were gorged with blood. (Ann. d'Hygiène, 1852, vol. ii, p. 423.)

Quantity required to destroy life.—With respect to this question, and *the period* at which the case proves fatal, there is no reason to suppose that the hydrochloric acid differs from the sulphuric and nitric acids in relation to these points. The cases that have hitherto occurred throw but little light upon these questions. The medical jurist must be content to draw an inference, the fairness of which cannot be disputed, when it is based upon the strong analogy which exists between the effects of this and the other two acids. Dr. Beck states that out of six cases of this kind of poisoning, five proved fatal. (Med. Jur. vol. ii, p. 448.) The facts at present before us are these: In one case, two ounces destroyed life in thirty-three hours; in a second, the same quantity killed a person in eight days; and in a third, a like dose proved fatal *in five hours and a half.* (See post, p. 269.) This, I believe, is the most rapidly fatal case on record. The smallest dose that appears to have destroyed life was about an *ounce.* The patient died in fifteen hours. But there have been three recoveries from a similar dose (one is reported, ante, p. 266; a notice of the second will be found in the Lancet for July 27, 1850, p. 113, and of the third, in Beck's Med. Jur. vol. ii, p. 449). In a case reported by Orfila, the dose was an ounce and a half, and this proved fatal in about eighteen hours. In one case (ante, p. 266), a much larger dose did not destroy life until after the lapse of eight weeks. If cases of poisoning by this substance were more frequent, it would, no doubt, be found that not only may death take place within a much shorter period, but that a much smaller quantity might prove fatal. The cases of poisoning by this acid, which I have found reported, have occurred in adults; some from accident, and others from suicide.

The following rapidly fatal case occurred to Mr. Crawford (Lancet, March, 1840): A woman, aged 40, took, in order to poison herself, two ounces of a mixture used by her husband for the purpose of browning gun-barrels. This mixture was stated to be composed of equal parts of tincture of steel and strong muriatic acid, with a few drops of a solution of corrosive sublimate. Soon after she had taken the liquid, she vomited. In about half an hour, she was seen by a druggist; vomiting had then ceased. She answered questions rationally, and was sensible until she died; but made no complaint of heat, or pain in the mouth, throat, or stomach. There was no thirst. The pulse could not be felt at the extremities, and the heart's action was very feeble. The muscles of the limbs felt hard, as if contracted. She died in about *five hours and a half* after taking the mixture. Two hours before death, the bowels were opened twice. On inspection, the stomach was found contracted, and its mucous membrane thrown into ridges and furrows. The ridges were of a brown color, as if charred; when the surface was scraped, it could be readily peeled off, and the part exposed showed numerous small black granules, which seemed to be nothing more than altered blood. The furrows were of a fine scarlet color. There was no perforation. Appearances similar to these were noticed in the upper part of the small intestines (duodenum and jejunum). The lower part of the gullet was charred, and its lining membrane was easily peeled off. There is no account given of the state of the mouth, throat, and air-passages. There is no doubt that the active agent and cause of death here was muriatic acid. An analysis of the mixture proved this, for it yielded an abundance of that acid, and a quantity of iron; but there was no trace of corrosive sublimate.

Treatment.—The same as in poisoning by sulphuric and nitric acids. (See ante, p. 240.) It consists in the free use of barley-water or linseed tea, with carbonate of soda.

In general the mineral acids are taken separately as poisons; but they may be taken in a mixed state; especially as some mixtures of this description are largely used in the arts. Thus, the Aqua Regia, a mixture of nitric and muriatic acids, is used for dissolving gold and platina; while the Aqua Reginæ, nitro-sulphuric acid, is employed for dissolving silver and separating it from plated articles. I have not been able to find in any work on toxicology, an account of a case of poisoning by the nitro-muriatic acid; but Orfila gives one case of poisoning by nitro-sulphuric acid. A man, aged 24, swallowed a mixture consisting of one ounce of strong nitric acid and two drachms of strong sulphuric acid. The usual symptoms followed, and he died in eight hours. The appearances, as might have been presumed from the relative quantities of the two acids taken, resembled those of nitric rather than of sulphuric acid. (Toxicologie Générale, i, 129.) There is but little doubt that nitro-muriatic acid would produce symptoms and cause appearances analogous to those described in speaking of muriatic acid. The mixed effects of nitric acid might be also perceptible. The quantity required to destroy life, and the period at which death will ensue, may be inferred from what has been already said respecting each of these acids.

CHEMICAL ANALYSIS.

The commercial spirit of salt has a deep lemon-yellow color. It may contain arsenic, antimony, iron, or common salt. It is not always so *concentrated* as to possess the property of fuming in the air; a property which of course depends on its strength, and therefore may be present or absent in any given specimen. A teaspoonful of this acid, having a specific gravity of 1·133, was found to weigh 66·4 grains, and a tablespoonful 265·6 grains. The liquid will be found highly acid: it tinges organic substances of a yellowish color, and corrodes them. 1. When boiled with copper, there is but little action; the acid is in part distilled over in vapor. By long boiling under free exposure to air, the copper acquires a pinkish or reddish hue (from a film of oxychloride). 2. The acid if moderately pure, may be boiled entirely away on pure mercury without being affected by the metal. These tests eminently distinguish the muriatic from the two preceding acids. 3. When boiled with black oxide of manganese, in fine powder, chlorine is evolved, known by its color, odor, and bleaching properties on litmus and other coloring matters. This last test is conclusive: there is no other acid which is thus affected by the peroxide of manganese. One drop of muriatic acid in a tube of very small bore, will give satisfactory results. The experiment may be performed on a small quantity in a flask as described under Nitric Acid (ante, p. 260), starch-paper dipped in solution of iodide of potassium being used as a test.

In the *diluted* state. When the acid is much diluted with water, the property of evolving chlorine with peroxide of manganese, is lost. In this case, there is one most satisfactory test for the presence of the acid —the *nitrate of silver*. This test gives, with the acid, a dense white clotted precipitate of chloride of silver. The precipitate thus formed acquires speedily a dark color by exposure to light; and it is known from all other white salts of silver, by the following properties: 1. It is insoluble in nitric acid. 2. It is very soluble in caustic ammonia. 3. It is insoluble in potash; but when boiled with a strong solution of potash, brown oxide of silver is produced. 4. When dried, and heated on platina, glass, or mica, it melts like a resin, forming a yellowish-colored sectile mass. Unless these properties be possessed by the precipitate, it is impossible to refer the action of the test to the presence of muriatic acid. The delicacy of the silver test is such, that it will easily detect the thirteen-hundredth part of a grain of muriatic acid in a minimum of water, and will form a perceptible opalescence (precipitate) when the acid is diffused through 290,400 times its weight of water.

Objections.—It may be objected to the application of the silver-test, that other acids form with it white precipitates, which might be mistaken for the chloride of silver. There are two common acids, both of them poisons,—namely, the prussic and the oxalic, which, in this respect, resemble the muriatic. The prussic acid would be immediately known by its odor, or by the effect of heat on the cyanide of silver. (See Prussic Acid.) The white precipitate produced by the test in oxalic acid, is known from the chloride by its entire solubility in nitric acid. The evaporation of a portion of the tested acid liquid, would moreover leave crystals of a solid acid.

The mixture of any simple acid, such as the acetic, tartaric, or citric, with a solution of common salt, might be pronounced to be muriatic acid from the action of the test, when in reality no free muriatic acid was present. A suspicion of this kind would naturally arise, if on evaporating a portion of the acid liquid, a large quantity of a solid white residue was obtained. The difficulty in such a case may be removed by resorting to the process recommended in speaking of sulphuric acid (ante, p. 243). If we take equal quantities of the acid liquid, and precipitate one portion entirely by nitrate of silver,—then evaporate the other portion to dryness, dissolve the dry salt (chloride) in water and precipitate this solution entirely by the test, it is obvious that if there be no free muriatic acid present, the precipitated chloride will have exactly the same weight in the two cases. The precipitate should in each case be well washed in water, acidulated with nitric acid. If free muriatic acid were present, the precipitate obtained in the former case would exceed in weight that obtained in the latter. As common salt is not very soluble in alcohol, the suspected liquid may be concentrated and treated with strong alcohol. This will dissolve any free acid and leave the chloride of sodium.

In liquids containing organic matter.—Such liquids will have a highly acid reaction. It might be supposed that the nitrate of silver would serve as a good trial test, but it is liable to be precipitated by most organic liquids, such as vinegar and porter, although no free muriatic acid may be present. This arises either from the presence of chlorides in liquid articles of food of this description, or from oxide of silver being itself precipitated by certain organic principles. In the last-mentioned case, the precipitate is known from the chloride by being redissolved in nitric acid. This test for muriatic acid cannot however be safely employed in the analysis of any liquid containing organic matter. Under these circumstances there are three methods of proceeding. 1. To distil the liquid at a low temperature in a retort fitted with a receiver. Any free muriatic acid will pass over, be condensed, and may now be safely tested. A mixture of sulphuric acid with a muriate in the liquid, would produce the same effect, and lead to error. The action of nitrate of baryta upon the acid organic liquid, would, however, show whether sulphuric acid were present or not. This process only answers when the muriatic acid is in moderately large proportion. If the quantity be small, none is obtained unless the distillation is carried to dryness; but then the process is open to objection. (See Ann. d'Hyg. Oct. 1842, ii, 339.) 2. We may evaporate to dryness a fractional portion of the organic liquid; calcine any residue, and observe whether on digestion in water and filtration, we obtain a solution of a chloride; if not, we must neutralize the acid organic liquid by adding carbonate of soda, then evaporate and incinerate the residue. In this way, we obtain all the muriatic acid contained in the liquid as chloride of sodium, and this may now be precipitated by the silver test. Should any chloride result from the first evaporation of the acid liquid, the quantity of muriatic acid thus obtained, must be deducted from that which results in the last-mentioned process.

Free muriatic acid may be separated from some organic substances, as well as from some alkaline salts, by digestion in alcohol. The alcoholic liquid may be filtered, and to the filtrate a strong alcoholic solution of caustic potash may be added. Chloride of potassium is formed and precipitated. This salt may be collected in a filter,—dissolved in distilled water, and tested. If carbonate of soda has been used in the treatment, the alcohol will not remove the chloride of sodium; but if magnesia has been used, even the chloride of magnesium will be dissolved and removed. Alcohol may thus enable us to ascertain whether any free muriatic acid remains in the parts submitted to analysis.

Vomited matters and contents of the stomach.—The process is the same in the two cases. The liquid should be separated from the solid portions by filtration through cotton or paper. If acid, we must proceed as directed in speaking of the analysis of an organic liquid. In giving evidence on this point, a witness may be asked, whether the natural secretions of the stomach do not owe their acidity to the presence of free muriatic acid. The experiments of Dr. Prout have proved that this is really the case: that the gastric secretions are acid, owing to the presence of free muriatic and acetic acids. An objection of this kind is answered by the facts, that the quantity of free muriatic acid, naturally contained in the gastric secretions, does not exceed the 1500th part by weight, *i. e.* it amounts to about five grains in sixteen ounces of liquid. (Prout.) This would only give a very feeble acidity, and but a trivial result with the test; whereas, the liquid may be intensely acid, and yield a large quantity of muriatic acid on being distilled. 2. The medical jurist would look for the characteristic symptoms and appearances, in the mouth, throat, and stomach, before he inferred that this mineral acid had been taken as a poison. If these are wanting, and the quantity of free muriatic acid is but small, then there would be no evidence of poisoning, so far as chemical analysis is concerned. A mixture of vinegar and salt might be easily mistaken for muriatic acid in an organic liquid, but no muriatic acid would be obtained on distillation, and no additional quantity of chloride would be obtained on neutralization by carbonate of soda and incineration. As organic matter holds muriatic acid with strong affinity, Orfila has recommended that it should be precipitated by a strong solution of tannic acid, before distillation is resorted to. The liquid for examination may be *neutral*, owing to the administration of antidotes. The muriatic acid may have been neutralized by carbonate of soda or magnesia. This would be discovered on evaporation, and the quantity of resulting alkaline chloride would indicate the quantity of muriatic acid. But to any inference of this kind, there are very strong objections. If the quantity of chloride of sodium is *small*, the results may be referred to that portion of salt which always exists naturally in the gastric and other secretions; if *large*, the chloride of sodium is so common an ingredient in most kinds of food, that its presence in the contents of a stomach, even in large quantity, might *cæteris paribus*, be fairly ascribed to this source. On the whole, then, it is clear that the chemical evidence in poisoning by muriatic acid must fail, unless the acid is discovered in large quantity and in a free state, in the contents of the stomach; or unless there is at the same time corroborative evidence of poisoning from symptoms and appearances. It need hardly be observed, that owing to violent vomiting or medical treatment, all traces of the acid may have disappeared from the stomach, notwithstanding the person may have died from its effects. With one alleged exception, muriatic acid has not been found in the stomach in the few cases of poisoning by it which are on record. Dr. Collas's case furnishes the exception here referred to (ante, p. 267), but the process pursued for its detection appears to me to have been unsatisfactory and inconclusive. The stomach and gullet were macerated in distilled water for sixteen hours: the liquid thus obtained was not acid, but gave a precipitate with nitrate of silver (alkaline chloride). A fourth part of it, with a fourth part of the stomach, being evaporated to dryness, was submitted to distillation in an oil bath with a small quantity of sulphuric acid. The gases and vapors thus evolved, precipitated nitrate of silver. The liquid taken from the stomach gave an abundant precipitate with nitrate of silver after distillation with sulphuric acid. Magnesia and soap had been given to the deceased. This might account for the absence of acidity; but the chemical results may have been owing to the presence of common salt in the stomach (Ann. d'Hygiène, Janvier, 1858, p. 216). The analyst must remember, that in examining the stomach of a person poisoned by this acid, he may discover traces of arsenic or antimony,—the commercial acid containing these substances as impurities.

Hydrochloric acid has not been used for the purpose of murder in this country, but in France there have been at least two trials for murder by this poison, *i. e.* in 1839 and 1847 (Orfila, Toxicologie, vol. i, p. 216). In a criminal case tried in Belgium, the acid was given with a view to procure abortion (Galtier, vol. i, p. 217). In these cases the evidence respecting the presence of poison in the bodies failed, although the appearances left no doubt that a corrosive acid had been taken. The analysts appear to have relied on the effect of nitrate of silver as a test, without having had due regard to the presence of alkaline chlorides in the stomach. On this account the chemical evidence was rejected as unsatisfactory, but as in the abortion-case the female did not die until after the lapse of two months, it would have been something unusual had any of this poison remained in the body. (The reader will find a report of these cases in Flandin's Traité des Poisons, vol. ii, pp. 482, 491.)

On solid organic substances.—Chemical evidence may be obtained from this source, when other sources fail. In Mr. Quekett's case (ante, p. 267), no muriatic acid was found in the stomach; but the nature of the poison was accurately determined by examining a piece of the deceased's waistcoat, on which some of the acid swallowed had become accidentally spilled. By digesting the stuff in warm distilled water, a highly acid liquid may be obtained on filtration, which, if muriatic acid be present, will yield, with nitrate of silver, a white precipitate, possessing all the properties of chloride of silver. The spots produced on black cloth by the strong acid are at first of a bright-red, but in ten or twelve days they change to a red-brown. Hence it will be perceived that this acid differs from the others in the effect produced on black cloth. Sulphuric and nitric acids produce brown and not red stains, the stain from the former acquiring a red fringe only after some days. An unstained portion of the cloth should always be examined by way of comparison. I have remarked that the red color produced by muriatic acid in black cloth is removed by boiling water, the cloth becoming black, but again on drying acquiring a red-brown color. The diluted muriatic, like the diluted sulphuric and nitric acids, produces at once red stains on black cloth.

If muriatic acid has been used in the erasure of writing ink for the purposes of *forgery*, its presence in the paper may be detected by a similar process. Supposing that there should be no free acid in the paper, the addition of ferrocyanide of potassium (by producing Prussian blue) will show that a soluble salt of iron (sesquichloride) has been diffused through the substance of the paper. A man of the name of Hart was tried at the Central Criminal Court, Dec. 1836, on a charge of forgery, under the following circumstances. The prisoner received a blank acceptance for £200, and afterwards erased the figure 2 by an acid, and substituted the figure 5. The witness who gave chemical evidence on this occasion, deposed that some acid had been used to effect the erasure, but he could not ascertain its nature. He suspected that it must have been either the muriatic or oxalic acid, probably the former. Counsel ingeniously objected to the evidence, that chloride of lime was used in the manufacture of the paper and might account for the results obtained by the tests; but in answer to this, it was properly stated, that the chloride was entirely removed by subsequent washing. If any acid liquid were obtained from a stain on paper under these circumstances, the muriatic would easily be known from the oxalic acid by the fact that the chloride of silver is not soluble in nitric acid, while the oxalate of silver is soluble in it.

Quantitative analysis.—This may be performed by estimating the quantity of muriatic acid by the quantity of chloride of silver obtained from the whole, or a fractional part, of the liquid subjected to analysis. A teaspoonful (=66·4 grains) of an acid having a sp. gr. of 1·133, gave 64 grains dry chloride of silver (=16·3 grains of dry hydrochloric acid gas). Such an acid would therefore have a strength of about 24 per cent. The *diluted* muriatic acid of the Pharmacopœia contains one-fourth by measure of strong acid; its sp. gr. is 1·043.

PHOSPHORIC AND PHOSPHOROUS ACIDS.

Phosphoric acid, according to Orfila, possesses, in a toxicological view, properties somewhat analogous to those which have been described of the other mineral acids, but in a milder degree. Nothing is known concerning its action on the human subject, as there is no instance recorded of its having been swallowed as a poison. It has been stated that the poisonous properties of phosphorus are owing to its conversion to this acid in the body; but reasons will hereafter be assigned to show that this is not sufficient to explain the facts. In their knowledge of the *symptoms* and *appearances*, toxicologists have nothing to guide them, therefore, but the results of a very few experiments on animals; and these are of a very conflicting nature.

Orfila gave to a dog *twenty-five grains* of the (anhydrous) phosphoric acid, dissolved in little more than its weight of water. In two minutes the animal vomited a reddish-colored mucous liquid, and the vomiting was repeated four times within the first hour. In two hours it appeared to suffer from pain in the throat, and made ineffectual attempts to vomit. On the following day the animal was depressed, and could neither stand nor walk. It died twenty-three hours after taking the acid. The mucous membrane of the stomach, near the pylorus, as well as that of the duodenum, was of a deep red color. According to Schuchardt, the acids of phosphorus have not the same kind of action as phosphorus itself, and when given in doses which would be poisonous with phosphorus, they produce no effect. The acids, however, were not given in a concentrated state. (Brit. and For. Med. Rev. vol. xix, 1857, p. 506.) In an experiment, performed by Dr. Glover, *fifty grains* of glacial phosphoric acid were given to a rabbit, dissolved in two fluid-drachms of water, but the dose produced no effect. (Ed. Med. and Surg. Journ. lviii, p. 121.) It can hardly be said, from the results of these experiments, that there is any analogy between the action of this and the other mineral acids, except when the latter are so much diluted as to have their corrosive properties entirely destroyed. Phosphoric acid appears to act only as an irritant, even when highly concentrated; but Dr. Glover's experiment shows that it does not possess any active properties. At least it would be necessary to give it in a considerable dose, in order to produce any well-marked effects.

Phosphorous acid.—In an experiment, performed by Hunefeld, and quoted by Orfila, twenty-one grains of phosphorous acid (*Phosphoriger Säure*) were given to a rabbit. For about an hour the animal appeared uneasy, and refused its food, but in a short time it completely recovered. After the lapse of twenty-four hours, sixty-two grains of the acid, dissolved in a small quantity of water, were given to it. Respiration became difficult—the animal was very uneasy: in ten or twelve hours it vomited a bloody fluid, and died in slight convulsions. The mucous membrane of the stomach, at the greater end, was of a red-brown color. This organ contained only a small quantity of phosphoric acid. The abdominal viscera were healthy, and no trace of the poison was found in them. The heart and lungs were gorged with blood,—the brain was healthy. The odor of phosphorus was not perceptible in any part of the body: but the urine was strongly impregnated with phosphoric acid,—a fact proved by adding to it ammonia and the sulphate of magnesia. (Toxicologie, vol. i, 177.)

Mr. Groves states that he has given to dogs doses of phosphorous acid varying from 9 to 22 grains. He experimented on six dogs: the gullet was tied after the injection of the diluted acid into the stomach, and the animals lived six, eight, or even nine days after the injection of the acid. The eventual death of the dogs was assigned to prolonged abstinence, and the results of the injuries consequent on tying the gullet. (Pharmaceutical Journal, April, 1858, p. 510.)

According to the experiments of Drs. Weigel and Krug, pure phosphoric acid has not any irritating action on the coats of the stomach, if given in an ordinary dose. When applied to the mucous membrane of the stomach of rabbits, it did not leave any sensible traces of corrosion. If, on the contrary, the same dose of phosphoric acid, containing only a tenth part of phosphorous acid, were given, the animals perished after some hours; and the mucous membrane of the stomach presented traces of gangrenous inflammation, which they ascribed to the peroxidation of the phosphorous acid. (Journal de Chimie Médicale, Mai, 1845, p. 288.) From these experiments, it would appear that phosphorous acid is far more powerful than phosphoric acid; and, admitting their correctness, the activity of phosphorus as an irritant poison may be probably due to its becoming, in the first instance, converted to phosphorous acid. Phosphoric acid, in a diluted state, is one of the medicinal compounds of the London Pharmacopœia. It contains 8·7 of phosphoric acid and 91·3 parts of water by weight. Its sp. gr. is 1·064. It is used as a tonic in doses of from twenty minims to one drachm.

Chemical analysis.—*Phosphoric acid* in solution. But one test is required, *i. e. Nitrate of silver.* This gives a milky opacity with phosphoric acid,—which is changed to a light yellow precipitate on the addition of a few drops of a weak solution of ammonia. The phosphoric might, in this respect, be confounded with arsenious acid, but it is easily known from this poison: 1. By its giving no deposit on copper when boiled with muriatic acid; and, 2. No yellow precipitate when treated with sulphuretted hydrogen gas. 3. It does not precipitate albumen, except when it has been brought to the solid or glacial state by evaporation. For phosphoric acid, in the *solid* state, or as it is procured by digesting a suspected powder in nitric acid and evaporating to a syrup, see PHOSPHORUS, post.

CHAPTER XVIII.

POISONING BY OXALIC ACID—SYMPTOMS AND EFFECTS—APPEARANCES AFTER DEATH—EXPERIMENTS ON ITS ALLEGED CORROSIVE ACTION ON THE STOMACH—PERFORATION—FATAL DOSE—RECOVERY FROM LARGE DOSES—PERIOD AT WHICH DEATH TAKES PLACE—TREATMENT—CHEMICAL ANALYSIS—TESTS FOR OXALIC ACID IN PURE AND MIXED LIQUIDS—OXALIC ACID IN ORGANIC SUBSTANCES—ITS ALLEGED PRODUCTION—POISONING BY THE RHUBARB OR PIE PLANT—TARTARIC ACID.

OXALIC ACID.

OXALIC ACID is one of the most powerful of the common poisons; but its use as a poison is almost entirely confined to this country. Cases of poisoning by it are generally the result of suicide or accident. In the Coroners' return for 1837–8, there were nineteen cases of poisoning by this substance, out of which number fourteen were the result of suicide. It is singular, also, that the greater number of these cases occurred in the County of Middlesex. Accidental poisoning by oxalic acid has arisen from its strong resemblance to Epsom salts. It is not often that we hear of its being used as a poison for the purposes of murder. Its intensely acid taste, which could not be easily concealed by admixture with any common article of food, would infallibly lead to detection long before a fatal dose had been swallowed. I have known several trials to take place for attempted poisoning by oxalic acid,—in two the vehicle selected for its administration was coffee or tea, and in one, the poison was powdered and mixed up with brown sugar to conceal the taste. (Reg. v. Dickman, Central Criminal Court, February, 1845.) In another, buttermilk is supposed to have been the vehicle of the poison.

SYMPTOMS.—In some cases of poisoning by this substance, death has taken place so rapidly that the person has not been seen alive by a medical practitioner. If the poison is taken in a large dose, *i. e.* from half an ounce to an ounce of the crystals dissolved in water, a hot burning acid taste is experienced in the act of swallowing, extending downwards to the stomach; and vomiting occurs either immediately, or within a few minutes. There is also a sense of constriction in the throat, almost amounting to choking or suffocation. Should the poison be diluted, there is merely a sensation of strong acidity, and vomiting occurs only after a quarter of an hour or twenty minutes. In some instances, there has been little or no vomiting; while in others, this symptom has been incessant until death. In one case, where the poison was much diluted, vomiting did not occur for seven hours. (Christison, op. cit. p. 221.) The vomited matters are highly acid, and have a greenish-brown or almost black appearance; they consist chiefly of mucus and altered blood. In a case which occurred to Dr. Geoghegan, they were colorless (Med. Gaz. xxxvii, 792); and in another, fluid blood of a bright arterial color was vomited after some hours. (Provincial Journal, June 25, 1851, p. 344.) There is at the same time a burning pain in the stomach with tenderness of the abdomen, followed by a cold clammy perspiration and convulsions. In a case that occurred in Guy's Hospital, in May, 1842, in which about two ounces of the poison had been swallowed, there was no pain. Urgent vomiting and collapse were the chief symptoms. There is in general an entire prostration of strength, so that if the person be in the erect position, he falls; there is likewise unconsciousness of surrounding objects, and a kind of stupor, from which, however, the patient may be without difficulty roused. Owing to the severity of the pain, the legs are sometimes drawn up towards the abdomen. The pulse is small, irregular, and scarcely perceptible; there is a sensation of numbness in the extremities, and the respiration, shortly before death, is spasmodic. The inspirations are deep, and a long interval elapses between them. In one case the patient was found insensible, and the jaws spasmodically closed. Such are the effects commonly observed in a rapidly fatal case.

The symptoms have been described as occurring *immediately* in this form of poisoning. This may give rise to an important question. The effect on the tongue and throat, as well as on the stomach, cannot be concealed, even if the vomiting and pain should not occur for some time; but in the case of children there may be a difficulty in drawing a conclusion. In Reg. v. Cochrane (Liverpool Summer Assizes, 1857), a woman was indicted for administering to two of her children, oxalic acid in half a pint of buttermilk. They were found dead in two hours, under circumstances of great suspicion: the body of one was cold. The room was full of smoke, owing to its having apparently been set on fire. The bodies were separately examined by different surgeons: one came to the conclusion, that the child whose body he had examined, had died from suffocation, the other, that the second child had died from corrosive poison. The mucous coat of the stomach in the child was inflamed and softened, so that it readily gave way. There were two brownish-colored patches, and the submucous coat was exposed. The stomach contained a fluid which, when examined by Dr. Edwards, yielded oxalic acid, to the amount of about forty-two grains. In the stomach of the other child, supposed to have been suffocated, twenty grains were found. There is no evidence that the children had vomited, and it is not improbable that death took place rapidly. The woman was acquitted. There was a difficulty in reference to the administration of the poison in this case, which has not before presented itself. A large quantity of the acid must have been dissolved in half a pint of buttermilk, to have destroyed these children, and have left so large a residue in the stomach. The children (æt. six and four respectively) are supposed to have swallowed this intensely acid liquid without difficulty or complaint and without any of the usual symptoms being produced!

Should the patient survive the first effects of the poison, the following symptoms appear: there is soreness of the mouth, constriction in the throat, with painful swallowing,—tenderness in the abdomen with irritability of the stomach, frequent vomiting, accompanied by purging. The tongue is swollen, there is great thirst. A patient may slowly recover from these symptoms.

In a case reported by Mr. Edwards, the patient, a female, lost her voice for eight days. In a former edition of this work, I treated it as doubtful whether the loss of voice in this case depended on the action of the poison. A case has, however, since occurred to Mr. T. W. Bradley, from which it may be inferred that a loss of voice may result from the direct effect of oxalic acid on the nervous system. A man swallowed a quarter of an ounce of the acid, and suffered from the usual symptoms in a severe form. In about nine hours, his voice, although naturally deep, had become low and feeble. This weakness of voice remained for more than a month, and its natural strength had not returned even after the lapse of nine weeks. During the first month there was numbness, with tingling of the legs. (Med. Times, Sept. 14, 1850, p. 292.) The occurrence of this sensation of numbness, and its persistence for so long a period after recovery from the symptoms of irritation, clearly point to a remote effect on the spinal nervous system. Spasmodic twitching of the muscles of the face and extremities have also been observed in some instances. (See Lancet, March 22, 1851, p. 329.)

From Dr. Christison's experiments, it would appear that this acid is still a poison, even when so diluted as to lose all its irritant and corrosive properties. It thus differs from the mineral acids. The effects vary according to the dose. In a large dose, but much diluted, the poison, he considers, destroys life by producing paralysis of the heart. When the dose is diminished, the spinal marrow is affected, and tetanus is one of the symptoms. When still less, but enough to prove fatal, the poison acts like a narcotic, and the animal dies as if destroyed by opium. (On Poisons, 219.) Dr. Pelikan, Professor of Medical Jurisprudence at St. Petersburgh, informs me that these variable modes of operation on animals, as a result of dilution, are not in accordance with his observations.

APPEARANCES AFTER DEATH.—The lining membrane of the mouth, tongue, throat, and gullet is commonly white and softened, but often coated with a portion of the dark-brown mucous matter discharged from the stomach. Sometimes the membrane has presented a bleached appearance. The *stomach* contains a dark-brown mucous liquid, often acid, and having almost a gelatinous consistency. On removing the contents, the mucous membrane will be seen pale and softened, without always presenting marks of inflammation or abrasion, if death has taken place rapidly. This membrane is soft and brittle, easily removed, and presents that appearance which we might suppose it would assume, if it had been for some time boiled in water. The small vessels are seen ramifying over the surface, filled with dark-colored blood, apparently solidified within them. The lining membrane of the gullet presents much the same characters. It is pale, and appears as if it had been boiled with water, or digested in alcohol; it has been found strongly raised in longitudinal folds, interrupted by patches where the membrane had been removed. In a case which was fatal in eight hours, the tongue was dotted with white specks; the gullet was not inflamed, but the stomach was much destroyed, and had a gangrenous appearance. With respect to the *intestines*, the upper portion of the canal may be found inflamed; but unless the case be protracted, the appearances in the bowels are not strongly marked. In a case of poisoning by this acid, however, which is recorded by Dr. Hildebrand, the mucous or lining membrane of the stomach and duodenum was very strongly reddened, although the patient, a girl of eighteen, died in three-quarters of an hour after taking one ounce of the acid, by mistake for Epsom salts. (Casper's Vierteljahrschrift, 1853, 3 B. 2 H. page 256.) In another case, in which two ounces of the acid had been taken, and death was rapid, the coats of the stomach presented almost the blackened appearances produced by sulphuric acid, owing to the color of the altered blood spread over them. It is worthy of remark that the glairy dark-colored contents of the stomach do not always indicate strong acidity until after they had been boiled in water. In protracted cases, the gullet, stomach, and intestines, have been found more or less inflamed.

I am indebted to Mr. Welsh for the particulars of a case of poisoning by oxalic acid which occurred in April, 1853. A woman, æt. 28, swallowed *three drachms* of the crystallized acid. She was found quite dead in *one hour* afterwards. On examining the body, both lungs were observed to be extensively congested, and the heart and large vessels were full of dark-colored blood. The stomach contained about three-quarters of a pint of dark-brown fluid, and its lining membrane was generally reddened. The other organs, excepting the brain, were healthy, and this presented appearances indicative of long-standing disease. There was serous effusion, with great congestion of the vessels. This case is remarkable from the smallness of the dose, the rapidity of death, and the well-marked redness of the mucous membrane of the stomach. The diseased state of the brain may have tended to accelerate death from the poison. In several instances there have been scarcely any perceptible morbid appearances produced.

Oxalic acid does not appear to have a strong corrosive action on the stomach, like that possessed by the mineral acids. It is, therefore, rare to hear of the coats of the organ being perforated by it. In experiments on animals, and in some observations on the human subject, I have found nothing to bear out the view that perforation is a common effect of the action of this poison. The acid undoubtedly renders the mucous coat soft and brittle, and it dissolves by long contact animal matter, which on analysis is found to be of a gelatinous nature. Its solvent powers on the animal membranes are not, however, very strong, as the following experiment will show. A portion of the jejunum of a young infant cut open, was suspended in a cold saturated solution of oxalic acid for six weeks. At the end of this time, the coats, which were white and opaque, were well preserved, and as firm as when they were first immersed, requiring some little force with a glass rod to break them down.

Dr. Christison refers to only one instance in which, after death from oxalic acid, the stomach was found perforated. Dr. Letheby has reported the following case. An unmarried female, æt. 22, of previously good health, swallowed one evening, in a fit of jealousy, a dose of oxalic acid (quantity not known), and the next morning she was discovered dead in her room. On inspection, the stomach was found much corroded and softened. The mucous membrane was much blanched, except in two or three places, where there were small black spots, as if blood had been effused and acted upon by the poison; and here and there a bloodvessel might be seen ramifying, with its contents similarly blackened. The coats of the stomach were so softened, that it could scarcely be handled without lacerating it. At the cardiac end the coats were of a pulpy or gelatinous consistency, and presented numerous perforations. The contents amounted to six ounces, and were of a dark color like porter, with but little solid matter. The liquid was strongly acid; and on being tested was found to contain about three drachms (180 grains) of oxalic acid. The softening effect here was probably due to long contact of a large quantity of the acid after death. (Med. Gaz. xxxv, 49.) In a case which occurred to Dr. Wood, in May, 1851 (Ed. Month. Jour. March, 1852, vol. xiv, p. 227), the stomach was found perforated. The deceased, a nursery-maid, æt. 27, was found dead on her right side, the knees drawn up to the abdomen, and the right arm was slightly extended. Dr. Wood was informed, that shortly before he was sent for she had vomited, and was unable to speak. (Some acid crystals were found in a saucer in the room.) It seems that while vomiting she fell on the floor senseless. On inspecting the body, thirty-five hours after death, there was a frothy liquid around the mouth, with minute acicular crystals. The tongue, pharynx, and gullet, had a bleached appearance; the gullet a bluish leaden-gray color, and the membrane was easily removed by a scalpel. The stomach presented a large irregular aperture at its upper and fore-part nearer the cardia than the pylorus. From this a dark gelatinous-looking matter, resembling coffee-grounds, escaped in abundance. The aperture, before handling, was of a size to admit the point of the finger; but it was enlarged by removal. It eventually presented the appearance of two large apertures separated by a narrow band. The internal surface of the stomach was occupied by the same grumous-looking fluid, and the mucous membrane presented an eroded appearance. The small intestines presented changes of a similar character. The larynx was filled with a frothy mucus. The heart and lungs were healthy. The left cavities of the heart as well as both lungs were gorged with blood. Oxalic acid was found in the vomited matters, and in the contents of the stomach. The acid had produced yellow spots on the cloth boots of the deceased.

Dr. Geoghegan has published a detailed account of the appearances met with in the stomachs of three persons poisoned by oxalic acid. The first was taken from the body of a young man, who must have died in about twenty minutes after swallowing the poison. The inspection was made the following day. The stomach contained about eight ounces of a dark-brown and viscid matter, resembling coffee-grounds, evidently largely impregnated with altered blood, and possessing an acid reaction. The mucous membrane of the larger end of the stomach was of a deep blackish-brown color, of variable intensity. The discolored condition of the membrane extended in narrow streaks into the body of the organ, where the lining membrane was otherwise of a uniform light purple-red color. Near the pylorus the membrane was translucent, and exhibited the dark ramiform vascularity of the submucous coat. The mucous membrane of the cardiac portion was soft and thin, detachable only as a pulp, and in parts eroded. In the body of the stomach the lining membrane was somewhat thickened, but less soft, removable in flakes of one-third of an inch; at the intestinal end not thickened, and yielding strips of one-fourth of an inch. The other organs were not examined, except the heart,—the blood in the right cavities of which was fluid; and the colon, the transverse arch of which was much contracted. In reference to the three cases, Dr. Geoghegan observes: Although in one of them, the contents, including no inconsiderable amount of acid, remained in contact with the coats of the organ, no perforation was observable, the solvent energies being diffused over a large surface. The dose was not ascertained in any of the cases. Oxalic acid and gelatin were discovered readily in the contents in all. The quantity of poison in the contents was, in the first case, about three to four grains; a much larger amount in the two latter, especially in No. 3. A comparison of these cases with those already on record gives as the ordinary appearances in the stomachs of persons who have succumbed to the influence of oxalic acid —1. Contents, of the color of coffee-grounds, consisting of altered blood and mucus, and separating into a supernatant fluid and insoluble deposit. 2. Softening of mucous membrane, with various shades of brown coloration, erosion, or gelatinization. 3. Brownish-black ramiform vascularity of the submucous tissue, owing to the imbibition of the acid contents. It is important to note in similar cases the coexistence of this latter condition with the state of the contents just described, as the ramiform vascularity, or diffuse brown discoloration, presents itself in many instances as a result of the action of lactic acid contained in the gastric juice. It appears evident that the fatal result in cases of poisoning by oxalic acid cannot be referred to the corrosion of the stomach as its chief cause, but rather to the contemporaneous and energetic action which it exerts in arresting the circulation. (Med. Gaz. xxxvii, 792.) The œsophagus, stomach, and bowels, have been chiefly examined in these inspections. Dr. N. Chevers describes the case of a man found dead at Mooltan, in September, 1853, with his right hand clasped over his stomach, showing a very sudden death. There were no marks of external violence; but the appearances of convulsions about the fingers and mouth. The stomach was found in a state of contraction from spasm. The mucous surface at the lower part was greatly and recently inflamed, and a minute quantity of oxalic acid was detected in the contents. The cerebrum and cerebellum were much congested. There was a slight effusion of serum beneath the pia mater (membranes), but no actual effusion of blood had occurred. (Medical Jurisprudence for India, p. 165.) The medical officer was inclined to refer death to congestion of the brain; but it was clearly due to oxalic acid,—the congestion being probably one of the effects produced by the poison. In a case in which I was consulted in December, 1854, a large dose of oxalic acid proved fatal in two hours. With the usual appearances in the stomach, the head and chest presented nothing unnatural. The heart contained dark fluid blood in all its cavities.

Fatal Dose.—A trial for murder by this poison took place in 1832, in which a question arose respecting the quantity of this poison required to destroy life. One witness deposed, that he though ten grains of the acid was sufficient; another said that it was not sufficient. The prisoner was acquitted. A question of this kind can only be solved by a reference to recorded facts; but unfortunately, in most cases, it has been impossible to determine exactly the quantity of poison taken. Oxalic acid, it is to be observed, presents some singular anomalies in its effects. In one case, a man swallowed, as nearly as could be ascertained, three drachms of the crystals: there was immediate vomiting, but no other urgent symptoms, and he recovered in a few hours. In a second instance, a woman took nearly half an ounce of oxalic acid—the usual symptoms appeared—she recovered in six days, and was able to leave the hospital. Mr. Semple met with a case, where a girl swallowed about two drachms of the poison dissolved in water. Vomiting occurred immediately. In about twelve hours the more urgent symptoms had disappeared; but there was still tenderness of the abdomen with irritability of the stomach. In the course of a few days the patient was quite well. In February, 1842, a case occurred at King's College Hospital, where a girl had swallowed two drachms of the acid, dissolved in beer. The only symptom from which she suffered on admission, was pain. She entirely recovered the next day. Dr. Babington of Coleraine has reported a case (Med. Gaz. xxvii, 870), in which a girl swallowed, by mistake, two scruples (*forty grains*) of the poison. Severe symptoms followed, chiefly marked by great irritation of the stomach. It was a week before this girl had recovered, and a much longer time elapsed before she was able to resume her duties. In these cases, it is to be observed, proper medical treatment was resorted to; and the effects of the poison may be therefore supposed to have been in a great degree counteracted. But this explanation is hardly sufficient to meet such cases as the following. A girl, æt. 15, swallowed two pennyworth (half an ounce) of oxalic acid, and she was not admitted into St. Thomas's Hospital until half an hour afterwards: a period had therefore elapsed, within which death has frequently taken place. When admitted, she complained of great heat, and a sense of burning about the throat and fauces, with a feeling of sickness at the pit of the stomach; she vomited a large quantity of bloody frothy mucus. The stomach-pump was used, and some prepared chalk in water was injected. After this she appeared sinking; collapse was coming on; the blood left the surface; the extremities were cold, and the pulse was hardly perceptible. Stimulants were given, and artificial warmth applied. The next day there was great soreness of the mouth and tongue, and the latter was swollen, red, and tender; skin hot; tenderness on firm pressure of the stomach. In a few days she perfectly recovered. (Med. Gaz. i, 737.)

In the summer of 1846 I saw a similar case, in which a like quantity had been taken by a patient in Guy's Hospital; and here the extremities were cold; but there was little pain on pressure of the abdomen some hours after the poison had been taken. This female recovered. It is not improbable that idiosyncrasy may account for these anomalies: *i. e.* that certain constitutions are with difficulty affected by this poison. Two cases have occurred at Guy's Hospital, in each of which half an ounce of oxalic acid had been swallowed. Active treatment was adopted, and both patients recovered. When the dose is upwards of half an ounce, death is commonly the result; but one of my pupils informed me of a case in which a man recovered, after having taken *one ounce* of the crystallized oxalic acid; and Dr. Brush of Dublin has communicated to the Lancet, a case in which recovery took place perfectly after a similar dose of the poison had been taken. The acid was in this instance taken by mistake for Epsom salts. One ounce was put into a tumbler, and boiling water was poured on it at night. About half-past four in the morning, the patient, a man aged sixty, stirred up the liquid and swallowed the whole. Contrary to what has been hitherto observed, there was no *immediate* vomiting: the man, having discovered his mistake, tried to excite it, and only partially succeeded after the lapse of ten minutes. Warm water was freely given to him, and he ejected from his stomach dark clotted blood mixed with mucus. The usual antidotal treatment was then resorted to, and the stomach-pump used. In two hours symptoms of collapse appeared. In about six hours the skin had regained its warmth; but there was no pain in the stomach or any part of the abdomen. The secondary symptoms were a burning sensation in the mouth and throat, great difficulty in swallowing, thirst, acid eructations, and drowsiness; and these symptoms continued for two or three days. Vomiting and irritability of the stomach remained until the sixth day, but from this time the recovery was rapid; and in about eighteen days all unfavorable symptoms had disappeared. (Lancet, July 11, 1846, 39.) In the same journal is reported another case of recovery after an *ounce* of the acid had been swallowed. The man, it is stated, was not seen until fourteen hours after he had taken the poison; and he had, in the meantime, travelled a distance of ten miles to Dublin. He had immediately taken warm water. On his arrival in Dublin, magnesia and rhubarb were given to him. He complained of a burning sensation in the throat and gullet; his tongue was coated; his pulse small, quick, and wiry. There was anxiety of countenance, with complete prostration of strength. The palate was blistered, and the throat was highly inflamed; there was tenderness of the stomach with vomiting of a dark substance mixed with blood. The man ultimately recovered, but for a long time afterwards he complained of a sense of constriction in the œsophagus. (Lancet, Sept. 13, 1845, 293.) The reporter of this case states, that the quantity of the poison actually taken exceeded an ounce. (See also a case by Mr. Allison, Lancet, Nov. 2, 1850, p. 502, and one by Dr. Barham, Prov. Med. Journal, Oct. 6, 1847, p. 544.)

According to the experiments of Mitscherlich, two drachms of this poison killed a rabbit in a quarter of an hour, and half a drachm killed another in half an hour. Fifteen grains produced general disturbance of the functions, but did not prove fatal. It is strange that this experimentalist should assert that oxalic acid does not produce inflammation of the intestinal canal. There are several cases here recorded which prove that this is a mistake—another instance of the fallacies of "animal" experience. The smallest fatal dose of this poison yet recorded, is *one drachm* or sixty grains. This fell under the observation of Dr. Barker of Bedford. (Lancet, Dec. 1, 1855.) He ascertained, on inquiry, that a boy, æt. 16, bought half an ounce of oxalic acid; he took about a quarter of it, eating it as a dry solid, and threw away the remainder. He was found in about an hour insensible, pulseless, and with his jaws spasmodically closed. He had vomited some bloody matter; his tongue and lips were unusually pale, but there was no excoriation. He died within nine hours after taking the poison. In Mr. Welch's case (supra) three drachms destroyed life in an hour.

It may be proper to state, that this poison is retailed to the public at the rate of from a quarter to half an ounce for one penny or twopence, and one ounce for twopence or fourpence.

PERIOD AT WHICH DEATH TAKES PLACE.—Equal quantities of this poison do not destroy life within the same period of time. In two cases in which about two ounces of the acid were respectively taken, one man died in twenty minutes,—the other in three-quarters of an hour. Dr. Christison mentions an instance in which an ounce killed a girl in thirty minutes; and another in which the same quantity destroyed life in *ten minutes*. Dr. Ogilvy of Coventry has reported a case of poisoning by oxalic acid, in which it is probable that death took place within *three minutes* after the poison had been swallowed. The sister of the deceased had been absent from the room about this period, and on her return, found her dying. The quantity of poison taken could not be determined. The only other remarkable circumstance in the case was, that the coats of the stomach were so softened, that on an attempt being made to remove the organ, they were lacerated by the weight of the contents. The intestines and left lobe of the liver were also found softened, as if by transudation. This is the most rapidly fatal case on record. (Lancet, Aug. 23, 1845, 205; and Med. Gaz. xxxvi, 831.) Dr Iliff has communicated to me the particulars of a case in which the wife of a druggist, who had taken a dose of oxalic acid, was found dead by the side of the counter within a few minutes after she had been seen living. The stomach contained a black viscid acid liquid. The mucous membrane was not destroyed, and there were no particular signs of inflammation. The veins were gorged with blood, which gave a peculiar appearance. The tongue was white, but neither the throat, gullet, or alimentary canal, presented any marks of inflammation. The vessels of the brain were turgid, and the pupils were dilated. When the dose of oxalic acid is half an ounce and upwards, death commonly takes place in an hour. There are, it must be admitted, numerous exceptions to this rapidity of action. Dr. Christison reports two cases, which did not prove fatal for thirteen hours: and in an instance that occurred to Mr. Fraser, in which only half an ounce was taken, the person died from the secondary effects in a state of perfect exhaustion, twenty-three days after taking the poison.

TREATMENT.—It is recommended that water should be sparingly given, as it is apt to lead to the more extensive diffusion and absorption of the poison. But, in some instances, water has been productive of great benefit, and has aided the efforts of the stomach to expel the poison by vomiting. (See the case by Dr. Brush, ante, p. 283.) The proper antidotes are chalk, compound chalk powder, magnesia, or its carbonate, made into a cream with water, and freely exhibited; or the bicarbonate of magnesia (fluid magnesia) may be at once employed. These remedies appear, from cases hitherto reported, to have been very efficacious when timely administered. A case, in which this treatment was successful, will be found reported by Mr. Tapson. (Med. Gaz. xxxi, 491.) The woman is said to have swallowed two ounces of the acid; but this is obviously a mistake. I agree with Dr. Christison in thinking, that it is more likely to have been two drachms.

A mixture of lime-water and oil may be advantageously employed. If much fluid has been swallowed, the stomach-pump may be resorted to, and the stomach well washed out with lime-water. The poison, in many instances, acts with such rapidity, as to render the application of these remedies a hopeless measure. The exhibition of the alkalies,—potash, soda, or their carbonates, should in all cases be avoided: since the salts which they form with oxalic acid, are poisonous as the acid itself. In the after-treatment (in the stage of collapse), warmth should be applied and stimulants administered.

CHEMICAL ANALYSIS.

In the simple state.—This acid may be met with, either as a solid, or in solution in water. *Solid oxalic acid:* It is seen more or less perfectly crystallized in four-sided prisms, in which respect it differs from other acids, mineral and vegetable. The crystals are unchangeable in air. They are soluble in water, and even in anhydrous alcohol; readily in alcohol at 0·815. The solubility in water is variously stated. I have found some specimens much more soluble than others; and the conclusion from the experiments which I have made is, that the acid is soluble in from twelve to fourteen times its weight of water. If there be any adhering nitric acid about the crystals, they are much more soluble. It is worthy of remark that this solution, unlike that of some other vegetable acids (tartaric and citric), undergoes no change or decomposition by keeping.

The crystals of oxalic acid are liable to be mistaken for those of two other salts, namely, the sulphate of magnesia (Epsom salts) and sulphate of zinc (white vitriol). The chemical differences are, however, well marked. Oxalic acid, when heated on platina foil, is entirely volatilized, or only a very slight residue of impurity is left, while the sulphates of magnesia and zinc are fixed. If the three substances be dissolved in water, it will be found that, while the solution of oxalic acid is intensely acid,—that of the sulphate of magnesia is neutral,—and of the sulphate of zinc very feebly acid. If a solution of caustic potash be added to the diluted solutions of the three bodies in water, those of magnesia and zinc yield white flocculent precipitates; that of oxalic acid remains unchanged. The most simple method of distinguishing them is by the taste. Oxalic acid is immediately indicated by the intense acidity of its solution.

For the further analysis of the acid, the crystals may be dissolved in distilled water; but should a suspected *solution* of the poison in water be presented for examination, it will be proper, after testing it with litmus-paper, to evaporate a few drops on a slip of glass, in order to observe, whether crystals be obtained. If there should be none, there can be no oxalic acid present. If *long and slender prisms* be procured, having an acid reaction, then it will be proper to proceed with the analysis of the solution.

TESTS.—1. *Nitrate of silver.* When added to a solution of oxalic acid, it produces an abundant white precipitate of oxalate of silver. A solution containing so small a quantity of oxalic acid as not to redden litmus-paper, is affected by this test; but when the quantity of poison is small, it would be always advisable to concentrate the liquid by evaporation before applying the silver test. The oxalate of silver is identified by the following properties: 1. It is completely dissolved by cold nitric acid, by which it is known from the chloride and cyanide. If collected on a filter, thoroughly dried, and heated on thin platina foil, it is entirely dissipated in a white vapor with a slight detonation. When the oxalate is in very small quantity, this detonation may be observed in detached particles on burning the filter previously well dried. 2. *Sulphate of lime.* A solution of oxalic acid is precipitated white by lime-water and all the salts of lime. Lime-water is itself objectionable as a test, because it is precipitated white by many other common acids—as the carbonic, tartaric, and phosphoric. The salt of lime, which, as a test, is open to the least objection, is the sulphate. As this is not a very soluble salt, its solution must be added in rather large quantity to the suspected poisonous liquid. A fine white precipitate of oxalate of lime is slowly formed. This precipitate should possess the following properties: 1. It ought to be immediately soluble in nitric acid. 2. It ought not to be dissolved by the tartaric, acetic, or any vegetable acid. Unless these precipitates be obtained, and their properties, as above described, determined, it cannot be said that oxalic acid is present in the solution.

Objections to the tests.—A liquid may be highly acid, yield crystals of oxalic acid on evaporation, and yet neither of the above-mentioned tests will act. This may happen when nitric acid is present in rather large proportion. As a matter of precaution in a doubtful case, the whole of the solution should be evaporated to crystallization, and the crystals dried and redissolved in water, before applying the tests. It may be urged that the nitrate of silver precipitates other acids. Thus it forms white precipitates with the muriatic and prussic acids, but these precipitates are insoluble in cold nitric acid, and do not detonate when dried and heated. The test gives only a slight turbidness with the tartaric and citric acids when highly concentrated, instead of the abundant milk-white precipitate which is produced in a solution of oxalic acid, even when considerably diluted. Besides, there is no common acid but the oxalic which is precipitated by sulphate of lime. One objection to *sulphate of lime* is, that it precipitates acid as well as neutral solutions of the salts of baryta and strontia; and an acid solution of chloride of strontium might somewhat resemble in this respect a solution of oxalic acid; but not to mention that there are various methods by which this kind of difficulty may be easily removed,—the sulphates of baryta and strontia are eminently distinguished from oxalate of lime by their rapid precipitation, and by their perfect insolubility in nitric acid.

It has been recommended to neutralize the oxalic acid by ammonia, or potash, before applying the tests. The tests then certainly precipitate oxalic acid more rapidly and abundantly. The objections, however, to the results are more numerous; and although these are easily susceptible of being removed in the hands of a practised toxicologist, it must be confessed that just doubts might often be entertained of the accuracy of the inference drawn from them by those who are not experienced in analysis. If the poison be already neutralized, as under the form of oxalate of ammonia, potash, or soda, we have no alternative but to apply them. But we must remember that, while the two tests above mentioned (nitrate of silver and sulphate of lime) are precipitated by very few *acids*, they are precipitated by numerous *salts;* and the precipitates possess properties, which it is sometimes difficult to distinguish from those of the oxalates of lime and silver. Thus the alkaline tartrates, citrates, carbonates, phosphates, pyrophosphates, borates when concentrated, and iodates, are precipitated by one or both of the tests, like the oxalates; and if chloride of calcium be used instead of the sulphate of lime, the alkaline sulphates would also be precipitated. It may be observed, however, that the recently precipitated tartrate and citrate of silver are distinguished from the oxalate, 1, by their being decomposed, when boiled, while the oxalate of silver retains its whiteness, and is not decomposed at a boiling heat, but, if oxalic acid be in excess, is readily dissolved, forming a clear solution. 2. The dried tartrate, heated on platina, burns without detonation; it becomes incandescent, and leaves a solid residue of metallic silver in a white fungous mass. The dried citrate partially decrepitates, and leaves a gray fibrous residue of metallic silver. Thus, then, these salts differ from the oxalate, which is entirely dissipated by detonation. Again, sulphate of lime does not precipitate an alkaline tartrate or a citrate, unless the salts are highly concentrated, while an oxalate is precipitated by the test even in the most diluted state. If, therefore, any doubt exists respecting the nature of the salt, it should be diluted with water before adding the test. The dried precipitates also differ. The oxalate of lime is silently converted, when heated on platina, to carbonate of lime or caustic lime, according to the temperature. The tartrate and citrate undergo partial combustion, leaving a gray or carbonaceous residue.

Other tests might be used—as, for instance, the chloride of gold, or the sulphate of copper, but they add no force to the evidence afforded by those above mentioned, and we may conclude that when we obtain from an acid solution, a solid acid substance crystallizing in well-defined slender prisms—these crystals remaining unchanged in air, being volatilizable without combustion, and giving, when dissolved in water, on the addition of nitrate of silver and sulphate of lime, the results above described, there can be no doubt that the substance is oxalic acid. Additional tests may or may not be employed, but any evidence, short of this, should not, it appears to me, be admitted to show the presence of the poison.

In liquids containing organic matter.—The process is the same, whether applied to liquids in which the poison is administered,—to the *matters vomited*, or lastly, to the *contents of the stomach.* Oxalic acid readily combines with albumen and gelatine, and it is not liable to be decomposed or precipitated by these or any other organic principles; it is, therefore, commonly found in solution in the liquid portion, which will then have a greater or less acid reaction. Should the liquid be very acid, we must filter it to separate any insoluble matters; should it not be very acid, the whole may be boiled, if necessary, with distilled water, before filtration is performed. A small quantity of the liquid may now be tested by a solution of *sulphate of copper.* This serves as a trial test; for oxalic acid is the only acid precipitated by it. If a greenish-white precipitate is formed either immediately or in a few minutes, oxalic acid may be present; but if none be formed, then either there is no oxalic acid present, or it is in very small quantity. This precipitate is not readily dissolved by strong nitric acid. Dr. Birkett has suggested an ingenious method of procuring crystals of oxalate of lime from liquids containing oxalic acid. (Med. Times and Gaz. April 28, 1855, p. 409.) On no account are the tests for oxalic acid to be employed in liquids containing organic matter, since nitrate of silver is easily precipitated by such matters, although none of the poison be present. The thick viscid liquid containing the acid, or the coats of the stomach itself (finely cut up) may be boiled with distilled water—the decoction filtered, evaporated to dryness, and the dry residue digested in alcohol (s. g. 0·815). Alcohol will dissolve the oxalic acid present, and this may be obtained crystallized on evaporation. The crystals drained from the colored liquid may be dissolved in water, and the solution, which will now have a strong acid reaction, may be filtered. To the filtered liquid, acidulated with a small quantity of acetic acid, acetate of lead should be added until there is no further precipitation; and the white precipitate formed,—collected and washed. The oxalic acid present in the liquid will exist in this precipitate under the form of oxalate of lead. There are two methods of separating oxalic acid from the oxalate of lead.

1. Diffuse the precipitate in water, and pass into the liquid, for about half an hour, a current of sulphuretted hydrogen gas, taking care that the gas comes in contact with every portion of the precipitate. Black sulphuret of lead will be precipitated; and with it commonly the greater part of the organic matter, which may have been mixed with the oxalate of lead. Filter, to separate the sulphuret of lead; the filtered liquid may be clear and highly acid. Concentrate by evaporation; the sulphuretted hydrogen dissolved in the liquid is thereby expelled, and oxalic acid may be ultimately obtained crystallized by evaporation on a glass slide, and examined by the microscope. If there were no oxalic acid present in the precipitate, no crystals will be procured on evaporation. If crystals are obtained, they should be purified by solution in alcohol and again dissolved in water. The aqueous solution will yield them sufficiently pure for determining their form,—the effects of heat as well as the other characters of oxalic acid.

2. The second process consists in boiling the precipitated oxalate of lead in water, containing a small quantity of diluted sulphuric acid (the proportion regulated by the quantity of precipitate) for half an hour. Sulphate of lead is formed, and oxalic acid is set free; this becomes mixed with any surplus sulphuric acid. Filter, and neutralize cautiously by ammonia: the liquid often becomes turbid from the precipitation of a small quantity of oxalate of lead suspended by sulphuric acid. Allow this to subside, and then test it with the nitrate of silver and sulphate of lime. The characters of oxalic acid, if it be present, are immediately brought out: the sulphate of ammonia, here formed simultaneously with the oxalate, does not in the least interfere with the application of the tests. I have used these processes in cases of poisoning, and have succeeded in detecting oxalic acid by them, in the contents of the stomach. Of the two, the first is the best adapted for obtaining crystallized oxalic acid; the second is the more expeditious for obtaining chemical evidence of the presence of the poison. The quantity of this poison found in the stomach after death is generally small. In one case, in which about an ounce and a half had been taken, and the person died in two hours, I found only thirteen grains. This is owing to the ejection of the greater portion by vomiting. In the case of Reg. v. Cochrane (Liverpool Summer Assizes, 1857), in which it was charged that two children, aged six and four years respectively, had been wilfully poisoned by their mother, it was stated by the medical witness, Dr. Edwards, that he found forty-two grains of oxalic acid in the stomach of the elder, and twenty grains in that of the younger child. It was not clearly established when or how this large quantity of poison could have been administered to the children, and the prisoner was acquitted. Dr. Letheby states that in his case he detected 180 grains (ante, p. 280).

Objections.—If, in the course of the analysis, acetate of lead should give no precipitate with the concentrated liquid, even when neutralised, then oxalic acid is not present in a quantity sufficient to be detected. If it should give a precipitate, still there may be no oxalic acid present. The medical jurist must remember, that the acetate of lead is precipitated by most kinds of organic matter, and by many mineral and vegetable acids and their salts. Thus, if he is operating on the contents of the stomach, the presence of Epsom salt (sulphate of magnesia), any alkaline sulphate, common salt (chloride of sodium), any tartrate, citrate, phosphate, or carbonate, would occasion a white precipitate with the acetate of lead. The presence of the sulphuric, muriatic, tartaric, citric, acetic, gallic, or tannic acid, either free or mixed with any of the above-mentioned salts, would produce the same result. A mixture of vinegar and salt, or of lemon juice and Epsom salts, would give an acid reaction, and be precipitated by acetate of lead like the oxalic acid. Common London porter is acid, and is precipitated by all the salts of lead. The answer to any objection of this kind is, that the analyst does not decide on the presence of oxalic acid from the effect of acetate of lead on the suspected liquid; but from the action of the proper tests for the poison on the acid substance separated by sulphuretted hydrogen from the precipitate which is formed by the acetate. This latter is not a test, but merely a means of separation to enable us to apply the other tests with safety. The nitrate of lead may be substituted for the acetate. It has this advantage, that, unlike the acetate, it is not so readily precipitated by the tartaric, citric, or gallic acid. Orfila condemns the plan here recommended for the separation of oxalic acid, because the same results would be obtained if binoxalate of potash or salt of sorrel were present in the organic liquid. Sorrel is much used in soup in France, but not, so far as I know, in England: hence, the objection is one of a national character. In order to remove the supposed difficulty, Orfila proposes that the evaporated organic liquid should be repeatedly digested with pure alcohol at common temperatures: this, according to him, will dissolve the oxalic acid, but not the binoxalate, and thus the two may be separated. (i, 190.) It would be indeed unfortunate for medical jurists if they were obliged to rely upon this process. Alcohol, as pure as it can commonly be procured, will certainly acquire an acid reaction when digested on binoxalate of potash. Hence, by relying on Orfila's process, the analyst would be deceived, and would pronounce oxalic acid, as such, to be present, when the deceased might only have partaken of the supposed sorrel soup. In order to meet this objection, it appears to me that we should inquire whether it is likely that the deceased had eaten sorrel? what were his symptoms before death? whether he died suddenly after partaking of some liquid or solid? whether there were any peculiar appearances in the throat, gullet, or stomach? whether the quantity of oxalic acid found in the stomach, would not be utterly incompatible with the hypothesis that it was due to the presence of salt of sorrel taken at a meal? A case of criminal poisoning by oxalic acid, is not likely to occur where an answer to one or more of these questions would not be readily obtained.

In order that the acetate of lead should act effectually, therefore, the solution of the poison must be concentrated to the smallest possible bulk. With less than three grains of oxalate of lead, it would be difficult to obtain a solution of the poison fitted for testing. This quantity would correspond to only about *one grain* of crystallized oxalic acid. Although the nitrate of silver is a delicate test, yet a certain weight of oxalate of silver must be obtained, in order to demonstrate the true nature of the precipitate. Less than a quarter of a grain would hardly give satisfactory results; but this quantity would correspond to about one-eighth of a grain of crystallized oxalic acid.

It is difficult to state the exact length of the period after death, at which we may expect to discover this poison in the contents of the stomach. Having on one occasion detected it in the contents of the stomach of a person who had been poisoned, I placed the liquid, containing much blood and mucus, aside for about five weeks during the summer. On reexamining it at the end of this period, it had become highly putrefied, ammonia had been formed, and not a trace of oxalic acid could be detected. Nevertheless, except when in very small quantity, and under exposure to extreme putrefaction, oxalic acid does not seem to be liable to disappear in contact with animal and vegetable substances. In one experiment forty grains of oxalic acid were added to six ounces of a mixture composed of gruel, porter, and albumen. The liquid was kept in a bottle loosely corked for a period of *twelve years.* After this lapse of time, the liquid was still acid, and oxalic acid was readily detected in it. The stomach, after death, may contain no traces of the poison. This will happen when the case is protracted,—vomiting has been urgent, or the stomach-pump employed. On the other hand, the poison may be present, but in an insoluble form, when lime or magnesia has been given as an antidote. White chalky masses may, in this case, be found adhering to the mucous surface of the stomach, or subsiding as a sediment in the liquid contents. The analysis required for oxalate of lime will answer for the oxalate of magnesia.

Oxalate of lime.—When oxalic acid exists in the stomach in an insoluble form, *i. e.* as white chalky masses of oxalate of lime, the following process may be adopted. The suspected oxalate, previously well washed, may be boiled for about twenty minutes, with an equal weight of pure carbonate of potash, in distilled water. A partial double decomposition takes place: the undissolved residue containing some carbonate of lime, and the liquid some oxalate of potash. The liquid may be filtered, neutralized by diluted nitric acid, and then tested with the tests already described for a soluble oxalate (ante, p. 286). If there be any desire to determine the nature of the alkali with which the oxalic acid is combined, some of the sediment obtained from the liquids, or scraped from the surface of the stomach, may be calcined on platina foil, when caustic lime or magnesia will be left, easily known from each other by their respective chemical characters.

Oxalic acid contained in organic substances.—I am not aware that oxalic acid has ever been distinctly found in a free state in the stomach and intestines, excepting in those cases in which it has been taken as a poison. Combined with lime, as oxalate of lime, it constitutes a well-known form of calculus found in the bladder, and is thus a product of peculiar chemical changes in the body. In certain diseased conditions of the system, or under the use of certain articles of diet, oxalate of lime is also freely passed, in a microscopically crystalline condition, with the urine. I once found traces of this salt in a calculus taken from the intestines, in a fatal case of perforation; but, although oxalic acid is thus produced in the human body, it appears to be immediately neutralized and rendered harmless by the base lime with which it meets in the secretions. In two fatal cases of perforation of the stomach, reported by Mr. Crisp, it is stated that the fluid which had escaped from the stomach contained a small quantity of oxalic acid. There was no satisfactory evidence as to the mode in which this acid found its way into the organ, whether by morbid secretion or otherwise: hence it does not appear to me that the alleged discovery of the acid in these cases can offer any objection to the statement here made.

Oxalate of lime, it may be remarked, is a large constituent of many dry lichens; it also enters into the composition of certain substances used for food or medicine; *e. g.* the leaves and root of the rhubarb-plant (*Rheum Rhaponticum*), sorrel, and turnips. The *stalks* of the leaves of the rhubarb-plant now constitute a very common article of food, and accidents are said to have arisen from its having been eaten in large quantity before the leaves were fully developed. The following case is quoted in the Medical Gazette, from the American Journal of Medical Sciences. A family of four persons, after eating very freely of the *leaves* of the domestic rhubarb or pie-plant, boiled and served as "greens," were all of them shortly afterwards seized with severe vomiting. In one of these persons the attack was followed by gastritis; but the others recovered soon after the vomiting. (Med. Gaz. vol. xxxviii, p. 40.) It is stated in the same journal, from an analysis by Dr. Long, that one pound of the plant yielded twenty-four grains of oxalic acid; but as this is combined with lime, it is not likely to exert a poisonous action. It is a question, whether, in certain stages of its growth, the oxalic acid may not be united to potash as binoxalate; for it is not probable that the quantity contained as oxalate of lime, in a moderate portion of the leaves of the plant, would cause gastritis.

The proportion of oxalic acid present in the combined state in the leaves and stalks of edible rhubarb has not been accurately determined, and it is probably liable to variation according to the stage of growth of the plant. The root has hitherto been chiefly examined. Mr. Quekett obtained from the root of Russian rhubarb from 35 to 40 per cent. of its weight of oxalate of lime. (Pereira, Mat. Med. vol. ii, pt. 2, p. 488.) This is an enormous proportion. According to another analysis, the proportion was 11 per cent. Dr. Schossberger, who has more recently investigated the subject, states that with a large quantity of sulphate of lime, he found but slight traces of oxalate of lime. (Pharmaceutical Journal, January, 1845.) Mr. Wilson has shown experimentally, that the oxalate of lime thus taken into the stomach is again rapidly excreted by the urine, and that it does not remain in the system. (Prov. Med. Jour. Sept. 2, 1846, p. 414; and Nov. 11, 537.) Admitting that oxalic acid combined with lime is a constituent of this plant in variable proportion, it is difficult to comprehend how this can form any objection to the inference from a medico-legal analysis of the contents of the stomach. Oxalate of lime can never be found in the stomach in a case of poisoning by oxalic acid, unless an antidote of lime has been given. But if an antidote has been used, there will be evidence from symptoms, and in this case the discovery of any portion of oxalic acid in the stomach after death, may perhaps not be at all material; the fact of poisoning would be sufficiently apparent from other circumstances. To give this objection any sort of force, it is necessary to suppose that a person, after having swallowed an enormous dose of rhubarb, or eaten an enormous quantity of the leaves, is by a mere coincidence seized with symptoms resembling those of poisoning by oxalic acid, and dies; further, that on inspection, no appearances indicative of the action of oxalic *acid*, are found in the throat, gullet, or stomach; and lastly, that the presence of a small quantity of oxalate of lime in the stomach, is of itself chemical evidence that the person has died from taking oxalic *acid!* Such a hypothetical case appears to me to carry with it its own refutation, in the facts which must necessarily accompany it. The employment of alcohol in the analysis of the contents of the stomach will allow us to separate any free oxalic acid from oxalate of lime, whether derived from the use of an antidote or from any other source.

The absorbed acid.—Oxalic acid is supposed to enter the blood, and give to it a dark brown color. In a case which proved rapidly fatal, where two ounces of the poison had been taken, I examined four ounces of blood taken from the vena cava; but not a trace of oxalic acid could be detected. Dr. Christison states that he did not succeed in detecting the poison in the blood when it had been purposely injected into the femoral vein of an animal which died in thirty seconds. Orfila was unable to obtain any traces of it from the livers or spleens of animals poisoned by the acid. (Op. cit. i, 247.) These negative results may be explained by supposing either that the poison is decomposed in the body, or that the means of separating it from organic compounds are not sufficiently delicate. In two cases, leeches, it is stated, have been killed by the blood drawn by them from persons who were at the time laboring under the effects of this poison. This seems to render it probable that the blood is poisoned, and, indeed, Orfila states that he has succeeded in detecting oxalic acid in the urine, although not in the solid organs. (Op. cit. i, 190.) According to Wöhler, it may be detected under the form of oxalate of lime in the urine of animals to which it has been administered. This fact should not be lost sight of by the medical jurist, as the oxalate of lime, although frequently found in certain states of disease, is not a normal constituent of urine. The microscope would here render great assistance, as the octahedral form of the oxalate of lime deposit is peculiar. It is probable that in acute cases, death is solely to be ascribed to the absorption of the poison and its peculiar action on the blood.

On solid substances.—When solid organic matters, such as cloth, paper, or linen, are impregnated with oxalic acid, proofs of this may be obtained by digesting the spots in water and applying the usual tests to the aqueous solution. Oxalic acid does not corrode these substances like a mineral acid, but it very slowly produces orange-colored spots with a red margin on black cloth, in which respect it differs from the other vegetable acids. This acid is sometimes used for removing writing-ink in cases of forgery, but in general, traces of iron are left in the paper, and the erasure may be detected by wetting the paper with ferrocyanide of potassium.

Quantitative analysis.—The quantity of oxalic acid present in a measured portion of any mixture, may be estimated by precipitating it entirely as oxalate of lead. Fifty grains of the commercial crystallized acid, when dissolved and precipitated by acetate of lead, gave 115 grains of dry oxalate of lead,—so that 100 grains of oxalate of lead would represent 43 grains of the crystallized acid. The weight of a teaspoonful of the crystals was found to be 76 grains; and half an ounce of the crystals (avoirdupois) corresponded to three teaspoonfuls by measure.

For an account of BINOXALATE OF POTASH, see post, p. 304.

TARTARIC ACID.

Tartaric acid has been generally considered not to possess any poisonous properties; but one case at least is on record, in which there was no doubt that this acid acted as an irritant, and destroyed life. The case referred to was the subject of a trial for manslaughter in the Central Criminal Court, in January, 1845. (Reg. v. Watkins.) The accused gave the deceased, a man, æt. 24, by mistake, *one ounce* of tartaric acid instead of aperient salts. The deceased swallowed the whole, dissolved in half a pint of warm water, at a dose; he immediately exclaimed that he was poisoned; he complained of a burning sensation in his throat and stomach, as though he had drunk oil of vitriol, and that he could compare it to nothing but being all on fire. Soda and magnesia were administered with diluent drinks. Vomiting set in, and continued until death, which took place nine days afterwards. On inspection, nearly the whole of the alimentary canal was found highly inflamed. The accused admitted that he had made a mistake, and tartaric acid was found in the dregs of the cup. The jury acquitted the prisoner.

Another case of poisoning by this acid, with a report of the results of analysis, has been published by M. Devergie. (Ann. d'Hyg. 1851, ii, 432.) This case gave rise to a controversy between the late M. Orfila and M. Devergie, the points in dispute relating chiefly to the processes for the detection of the acid in the stomach and tissues. (See Ann. d'Hyg. 1852, i, 199, 382, and ii, 230.)

Dr. Mitscherlich has performed with this acid a series of experiments on animals, which tend to prove that it is not an active poison. He found that while the animal was under the influence of the acid its breathing was accelerated, and it then became laborious and slow. Great debility was a prominent symptom, and soon ended in paralysis, death being preceded by slight spasms. He considered this acid to be less noxious than the citric. Half an ounce was administered to a small rabbit, and proved fatal in one hour; three drachms killed a similar animal in forty minutes; and two drachms, given to a middle-sized animal, produced no effects. In the fatal cases, it was not found to excite inflammation of the small intestines. Tartaric acid appears to enter into the blood, and to act by absorption, for Wöhler detected it as a tartrate of lime in the urine of animals to which he had administered it. (Med. Times, Sept. 1845, 341.) Dr. Christison states that he has given to cats one drachm of this acid in solution, without apparently producing any inconvenience to the animal! and that a surgeon of his acquaintance had known six drachms of tartaric acid to have been taken by an adult, in mistake for carbonate of potash, without exciting unpleasant symptoms. (On Poisons, 227.)

TREATMENT.—The same as in poisoning by oxalic acid.

CHEMICAL ANALYSIS.—Tartaric acid in powder is known by the following characters: 1. When heated on platina-foil it burns with a pale reddish-colored flame evolving a peculiar odor and leaving an abundant residue of carbon. 2. It forms an acid solution in water, which, when moderately concentrated, yields a granular precipitate with a few drops of caustic potash slowly added. (Bitartrate.) A little alcohol facilitates the precipitation. 3. When a few drops of the acid solution are evaporated on glass, it crystallizes in an irregular plumose form. 4. The solution is precipitated white by lime-water, when the latter is added in large quantity; the precipitate being immediately dissolved by an excess of the acid. 5. It gives no precipitate, or only a slight opacity with nitrate of silver (thus known from oxalic acid). 6. It is not precipitated by chloride of calcium. 7. When exactly neutralized by potash, and nitrate of silver is added, a white precipitate is formed, which is immediately blackened and reduced to the state of metallic silver on heating the liquid to 212°. 8. When the powdered acid is heated with strong sulphuric acid, it is blackened.

Organic mixtures.—If the acid be not discovered in the stomach in the state of powder or crystals, we may digest the contents in alcohol, in which the vegetable acid is quite soluble.

For an account of BITARTRATE OF POTASH, see p. 305, post.

CHAPTER XIX.

POISONING BY THE ALKALIES, POTASH, SODA, AND THEIR CARBONATES—SYMPTOMS—APPEARANCES—TREATMENT—ANALYSIS—AMMONIA AND CARBONATE OF AMMONIA (SAL VOLATILE)—CHEMICAL ANALYSIS OF AMMONIA.

POTASH AND SODA.

SYMPTOMS.—The symptoms produced by potash and soda, when taken in large doses, are so similar, that one description will serve for both. It must be observed, that cases of alkaline poisoning are extremely rare, and have been, I believe, hitherto the result of accident. The most common form in which these poisons are met with, is in the state of pearlash (carbonate of potash) and soap-lees (carbonate of potash or soda mixed with caustic alkali). The patient experiences, during the act of swallowing, an acrid, caustic taste: the alkaline liquid, if sufficiently concentrated, softens and corrodes the lining membrane. There is a sensation of burning heat in the throat, extending down the gullet to the pit of the stomach. Vomiting is not always observed; but, when it does occur, the vomited matters are sometimes mixed with blood of a dark-brown color, and with detached portions of mucous membrane: this effect depending on the degree of causticity in the liquid swallowed. The surface is cold and clammy: there is purging, with severe pain in the abdomen, resembling colic. The pulse is quick and feeble. In the course of a short time, the lips, tongue, and throat, become swollen, soft, and red.

Appearances after death.—There are strong marks of the local action of the poison on the mucous membrane of the mouth, throat, and gullet. This membrane has been found softened, detached, and inflamed in patches of a deep chocolate color,—sometimes almost black. The same appearance has been met with in the lining membrane of the larynx and trachea. The stomach has had its mucous surface eroded in patches, and there has been partial inflammation.

Dr. Barclay has reported a case of chronic poisoning by potash, which furnishes a good illustration of the after-effects and appearances caused by this poison. A woman, aged 44, was admitted into St. George's Hospital, May 2, 1853, about six hours and a half after she had swallowed a quantity of American potash,—probably a saturated solution of carbonate of potash (American pearlash). She had vomited immediately after taking it. The mouth and fauces were much corroded. There was burning pain in the throat and gullet, extending downwards to the stomach; but there was no tenderness on pressure. Two days after her admission, there was a little vomiting. The mucous membrane, so far as it could be seen, was destroyed; there was difficulty of swallowing, and occasionally pain after food had entered the stomach. In about a month there was frequent vomiting, with pain on pressure, and constipation; when food or medicine was taken, there was much pain in the stomach, and in a short time the food was ejected. As the case progressed, nothing could be retained on the stomach, and, shortly before death, the patient was supported only by nutritive injections. She died from starvation on the 8th July, about two months after taking the alkali. On inspection, the lower part of the gullet was found much contracted, the lining membrane entirely destroyed, and the muscular coat exposed. The external coats were much thickened. The cardiac orifice of the stomach, where the ulceration ceased, was considerably contracted. At the pyloric end, the mucous lining presented a large and dense cicatrix, obstructing all communication with the duodenum, except by an orifice no larger than a probe. The intervening portion of the stomach was healthy, as were also the large and small intestines. (Med. Times and Gazette, Nov. 26, 1853, p. 554.)

Orfila refers to two cases of poisoning by carbonate of potash, in each of which half an ounce of this substance was taken by mistake for aperient salts. The patients, two young men, recovered from the first effects, but ultimately died; the one three months, and the other four months, after the poison had been taken. The secondary fatal effects appear to be due to constant purging, great irritability of the stomach leading to incessant vomiting, or loss of the functions of this organ from the destruction of the lining membrane, and stricture either of the gullet or of the apertures of the stomach,—any of which causes may prove fatal at almost any period. A fatal case of stricture, produced by soap-lees after the lapse of two years and three months, is reported by Dr. Basham (Lancet, March 2, 1850). The constant use of the alkalies or their carbonates appears to be productive of latent mischief: yet the quantity which may be sometimes taken in divided doses, without destroying life, is enormous. Dr. Tunstall, of Bath, relates the case of a man who for eighteen years had been in the habit of taking bicarbonate of soda to remove dyspepsia. It is stated that, for sixteen years, he took *two ounces* of the bicarbonate daily! The man died suddenly, and, on examining the stomach, it was found to be greatly distended and extensively diseased,—conditions which were referred by Dr. Tunstall to the action of the carbonate of soda (Med. Times, Nov. 30, 1850, p. 564).

In a case which occurred to Dr. Deutsch (Berlin Med. Zeitung, 1857, No. 51), a man, æt. 55, drank by mistake a quantity of soap-lees, containing about 30 per cent. of caustic potash. It was calculated that the quantity taken must have contained half an ounce of potash, of which a fourth part was considered to have reached the stomach. He was seen almost immediately. The lining membrane of the mouth and throat had a bluish-red color, was separating in shreds, and easily bled on being touched. The man complained of an insupportable burning pain extending from the mouth to the stomach,—a nauseous taste and a sensation of constriction in the gullet. He could not swallow: any attempt at swallowing gave rise to constriction of the throat. Choking and an inclination to vomit existed, but he did not completely vomit. There was cold perspiration, with paleness of the face,—collapsed features, slight convulsions,—hiccough, and a rapid, small, thready pulse. The abdomen was distended and tender to the touch. The taking of liquids produced vomiting of bloody shreds of mucous membrane. In eight days the inflammation was reduced. After six weeks there was still difficulty of swallowing: this increased, and the man died from starvation twenty-eight weeks after he had swallowed the alkali. On inspection, the throat and upper part of the gullet presented nothing abnormal; but the gullet became thicker and more contracted as it descended to the stomach, so that the opening into the stomach would scarcely admit a crow-quill. This was owing to a thickening of the mucous membrane. The muscular coat had almost disappeared. The stomach was empty, small, contracted, and bloodless, but free from any organic changes (Med. Times and Gaz. May 22, 1857, p. 537).

Fatal dose and period of death.—The earliest fatal case which I have found reported, is that of a boy, who died in *three hours* after swallowing three ounces of a strong solution of carbonate of potash. In another case, which occurred in 1835, a child aged 3 years, took a small quantity of pearlash, which had deliquesced, and died in twenty-four hours. Death was caused in this instance by the inflammation induced in the larynx, causing an obstruction to breathing. In this respect, the caustic alkalies may destroy life like the mineral acids,—by the local effects on the air-passages. In an instance which was communicated to me, a lady swallowed, by mistake, one ounce and a half of the common solution of potash of the shops, which contains about five per cent. of caustic alkali. She recovered from the first symptoms of irritation, but died seven weeks afterwards, from pure exhaustion,—becoming greatly emaciated before her death.

Treatment.—We may administer freely, water containing acetic or citric acid dissolved,—lemon-juice, or the juice of oranges. Demulcent drinks, as albumen, milk, gruel, or barley-water, will also be found serviceable. The free exhibition of oil has also been found useful.

Chemical analysis.—Caustic potash and soda are known from their respective carbonates by giving a brown precipitate with a solution of nitrate of silver. The *carbonates*, on the other hand, yield a whitish-yellow precipitate. Caustic *potash* is known from caustic *soda* by the following characters. 1. Its solution, when not too much diluted with water, is precipitated of a canary-yellow color, by bichloride of platina. 2. It is precipitated in granular white crystals, on the addition of an excess of a strong solution of tartaric acid. This test answers better by adding the alkali gradually to the acid;—and by the addition of a little alcohol to the mixture. Caustic *soda* is not precipitated by either of these tests, which will serve equally to distinguish the salts of potash from those of soda, if we except the binoxalate and bitartrate of potash, which, from being but little soluble in water, are not precipitated. 3. If we neutralize the two alkalies by diluted nitric acid, and crystallize the liquid on a slip of glass, should the alkali be potash, the crystals will be in the form of long slender fluted prisms; if soda, of rhombic plates. 4. A fine platina wire may be dipped into the alkaline liquid, and then dried by holding it above the flame of a spirit-lamp. In this way, a thin film of solid alkali is obtained on the wire. On introducing this into the colorless part of the flame, if it be potash, the flame will acquire a rose or lilac color; if soda, a rich yellow color. This test applies to the salts of the two alkalies. Care must be taken that the platina wire is perfectly clean. When the quantity of alkali or alkaline salt is large, the experiment may be performed in a platina capsule, alcohol being added to the salt, and the mixture boiled.

The *carbonates* of potash are known from those of soda by the above tests. The *carbonate* is known from the *bicarbonate* of either alkali, by the fact that the former yields immediately a white precipitate, with a solution of sulphate of magnesia, while the latter is unaffected by this test until the mixture is heated. It is important for the analyst to remember that caustic potash and soda, their respective carbonates, and the sesquicarbonate of ammonia, are often contaminated with oxide of lead, and give a black precipitate with sulphuretted hydrogen or hydrosulphuret of ammonia. This happens whenever the solution of these salts have been kept in flint-glass bottles.

In liquids containing organic matter.—Such liquids are frothy: they possess an alkaline reaction, a peculiar alkaline odor, and are unctuous to the feel. Potash and soda soften and dissolve most kinds of animal and vegetable matter. They also act upon woollen articles of clothing. If the organic liquid be highly alkaline, and gives out no odor of ammonia, either by itself or on distilling a portion of it with caustic potash, the alkali may be either *potash* or *soda*, or their *carbonates*. The latter would be known by the liquid effervescing on adding a portion of it to an acid. The organic liquid may be evaporated to dryness, then heated to char the animal and vegetable matter, and the alkali will be recovered from it in the state of carbonate by digesting the residuary ash in distilled water. Any caustic alkali may be effectually separated by digesting the incinerated residue in pure alcohol. It has been also recommended to neutralize by muriatic acid, to evaporate, incinerate, and procure the alkali for analysis in the state of chloride. Mere traces of these alkalies furnish no evidence of poisoning, since all the animal liquids and solids yield soda, and many of them potash. In no case will the discovery of the alkalies be any proof of poisoning, unless the alkali be in *large* quantity, and the marks of its action be apparent in the throat and stomach.

If the alkali be *ammonia*, this will be announced by the odor, and it may then be obtained by distillation. If it be in small proportion, this can afford no evidence of poisoning; since many animal fluids contain ammonia, and in those which do not contain it, it is easily generated either by spontaneous decomposition, or sometimes even by the heat required for distillation with potash. Should the alkali be in large quantity, this is no evidence of poisoning by it, unless we, at the same time, discover obvious marks of its local action on the mouth, throat, gullet, and stomach.

Absorption.—According to the experiments of Orfila, potash is *absorbed* and conveyed into the blood. The alkali is eliminated by the urine, which is thereby rendered alkaline. When he gave about one drachm of potash to dogs, the presence of this alkali was detected after the lapse of six hours, in the liver, spleen, and kidneys. Owing to the solvent action of this poison on fibrine and albumen, the blood, although it may be darker in color, is never found coagulated in the vessels after death.

AMMONIA.

Vapor.—The vapor of strong ammonia is poisonous. It may destroy life by producing violent inflammation of the larynx and of the lungs. The vapor produces a feeling of choking with a sense of great heat in the throat: it appears to suspend the power of breathing, and the pain and heat in the throat remain for a long time. The vapor is often most injudiciously employed to rouse persons from a fit. A case is on record, of an epileptic having died, under all the symptoms of croup, two days after the application of the vapor of strong ammonia to his nostrils. A singular case of recovery from the poisonous effects of this vapor, by Dr. Souchard, will be found reported in the Annales d'Hygiène (1841, vol. i, p. 219).

Symptoms and appearances.—A strong *solution* of ammonia produces symptoms similar to those described in speaking of potash. The only difference observed is, that the sense of heat and burning pain in the throat and stomach is much greater. Cases of this form of poisoning are rare. Dr. Souchard relates an instance which occurred in France, where a boy, only 6 years old, poisoned his younger sister by pouring several teaspoonfuls of strong solution of ammonia down her throat. In the following instance of poisoning by ammonia, the exact quantity taken was unknown, but the solution was sufficiently strong to act chemically on the mouth. A woman, æt. 24, swallowed about half a wineglassful of a mixture containing a large quantity of strong solution of ammonia put into it by mistake. She immediately fell backwards in a state of insensibility, and appeared as if choked. When seen about six hours after the accident, she complained of severe burning pain down her throat and in the stomach, which was tender on pressure. There was great debility, the voice was reduced to a whisper, and the countenance expressed anxiety. There was also great difficulty of swallowing, the pupils were widely dilated, the breathing was difficult, the tongue coated with a white fur, painful, and tender; two or three patches of its mucous membrane had peeled off, and there were convulsive twitches of the right arm. Diluted vinegar and other remedies were employed, but the patient did not entirely recover from the effects until after the lapse of ten days. On the fifth day there were still great pain and tenderness in the abdomen. (Case by Mr. Wilkins, Lancet, April 4, 1846, p. 385.) A case is referred to in the Journal de Pharmacie (Oct. 1846, p. 285), in which from one to two drachms of ammonia, unknowingly administered, caused death. There was violent vomiting, with bloody purging; and, on inspection, blood was found effused in the intestines. There was also a remarkably fluid state of the blood in the body. In another instance, a man walked into a druggist's shop, and asked for a small quantity of ammonia to take some spots out of his clothes. The druggist poured about a teaspoonful and a half into a glass. The man suddenly swallowed it, and fell instantly to the ground. He soon afterwards died, complaining of the most excruciating pain. (Journal de Chimie Médicale, 1845, 531.)

In August, 1854, a woman swallowed ten drachms of a solution of ammonia (strength not stated); she threw the glass from her and rushed into an adjoining room. When seen by a medical man she was in a sitting position, having on her knees a basin containing a large quantity of stringy salivary fluid, with a few streaks of blood. The face was pale, the eyes were haggard and injected, the lips presented much swelling, and also redness, which extended to the mouth and throat. There was entire loss of voice. There was pain in the pharynx and stomach. The pulse was slow, and the limbs were cold. Some spoonfuls of vinegar were given, but were swallowed with difficulty. The pain in the stomach was severe, and was increased on pressure. A draught of cold milk, which happened to be at hand, was given to the woman, and produced relief. The loss of voice and the inability to swallow lasted three days; a large quantity of saliva with a bloody membrane was thrown off; the pain in the stomach continued. The patient recovered in a week. (L'Union Médicale, Fev. 19, 1857; Brit. and For. Med. Rev. 1857, vol. xix, p. 500.)

A man, æt. 40, had been in the habit of taking one drachm of solution of ammonia diluted with water to relieve his breathing. In June, 1857, he took into his mouth by accident an ounce of hartshorn spirit. He suddenly called for water and quickly ejected the fluid. He was immediately seized with intense burning pain and a feeling of suffocation. He thought that none of the fluid had reached the stomach. No antidote was administered. In two hours he was seen laboring under the following symptoms: countenance suffused,—lips livid,—breathing stridulous,—aspect anxious,—legs cold,—beating the bed-clothes with his hands (malleatio!), pulse 100,—the inside of the mouth, tongue, and throat, so far as could be seen, red, raw, and fiery-looking. There was pain referred to the larynx, but no pain in the stomach, even on pressure: there was no thirst. The symptoms of injury to the respiratory organs were the most marked. Under these he sank on the twentieth day from laryngismus stridulus. (Ed. Med. Journ. 1857, p. 236, case by Dr. Paterson.)

Serious injury to the organs of respiration is frequently the result of the local action of this poison, as in the following case, which was referred to me for examination by my colleague, Mr. Hilton, in May, 1857: A gentleman liable to attacks of fainting died in three days, after swallowing a quantity of liquid administered to him by his son. This liquid, which was at the time believed to be sal volatile, was, in fact, a strong solution of ammonia. The deceased complained immediately of a sensation of choking and strangling in the act of vomiting. Symptoms of difficulty of breathing set in, with other signs of irritation in the throat and stomach. The mucous membrane of the mouth and throat was corroded and dissolved; and it was evident that the liquid had caused great local irritation. The difficulty of breathing was such as to threaten suffocation, and at one time it was thought that an operation must be resorted to. The state of the patient, however, precluded its performance, and he died on the third day. On inspection, the viscera presented strong marks of corrosion. The covering of the tongue was softened and had peeled off; the lining membrane of the trachea and bronchi was softened and covered with layers of false membrane,—the result of inflammation,—the larger bronchial tubes were completely obstructed by casts or cylinders of this membrane. The lining membrane of the gullet was softened, and at the lower part, near its junction with the stomach, the tube was completely dissolved and destroyed. There was an aperture in the stomach in its anterior wall, about one inch and a half in diameter; the edges soft, ragged, and blackened, presenting an appearance of solution. The contents of the stomach had escaped. On the inside, the vessels were injected with dark-colored blood, and there were numerous small effusions of red blood in various parts of the mucous membrane. The coats were thinned and softened at the seat of the aperture. The blackened and congested appearance somewhat resembled that which is seen in poisoning by sulphuric or oxalic acid. The mucous matter on the coats of the stomach was feebly acid. No poison of any kind was found in the layer of mucus, or in the coats. There was not in any part the slightest trace of ammonia,—the poison which had caused the mischief. The deceased had lived three days: remedies had been used, and every trace of ammonia had disappeared. The immediate cause of death was an obstruction of the air-tubes, as a result of inflammation, caused by the irritant action of the poison. It was quite obvious that a quantity of the liquid had entered the windpipe. The perforation of the stomach had probably taken place shortly before death, or there would have been marks of peritonitis. The injury to the stomach and œsophagus would have been sufficient to cause death, even supposing that the liquid had not penetrated to the lungs.

Fatal dose.—The effects will depend more on the amount of injury to the air-passages and the stomach, than on the precise quantity taken. Death may be a very rapid result of the action of this poison.

The treatment in these cases must vary with the symptoms. Acetic acid and water, or milk with barley-water may be given if the power of swallowing should exist.

CARBONATE OF AMMONIA—HARTSHORN—SAL VOLATILE.

Symptoms and appearances.—The solution of this salt (sal volatile) is probably more active as a poison than is commonly supposed. The following case occurred in 1832: A man in a fit of passion swallowed about five fluid-drachms of a solution of sal volatile. In ten minutes he was seized with stupor and insensibility; but, upon the application of stimulant remedies, he recovered. He suffered for some time afterwards from severe irritation about the throat and gullet. Dr. Iliff met with the case of a boy, æt. 2 years, who swallowed about half an ounce of spirit of hartshorn. He immediately screamed, and was very sick; bringing up at first stringy mucus, at first of a light, and afterwards of a dark color. The lips were swollen—the breathing hard, hurried, and somewhat obstructed. There was perfect sensibility. The fore part of the mouth was but little affected; there was pain and difficulty in swallowing. The most urgent symptoms were connected with the lungs and air-passages. In three days the boy recovered. (Lancet, Dec. 1, 1849, p. 575.)

In a paper above referred to, Dr. Barclay relates the case of a female, æt. 19, who, while in a state of unconsciousness, was made to swallow a quantity of hartshorn. She felt a severe pain in the stomach immediately afterwards, and in about an hour she vomited some blood. This vomiting of blood continued for several days. These symptoms were followed by great irritability of the stomach, and the constant rejection of food. There was obstinate constipation of the bowels, with great emaciation and loss of strength. She died in about three months from the time at which she had swallowed the alkaline poison. On inspection, the gullet was found healthy; the orifice, at its junction with the stomach, was slightly contracted. The pyloric opening was contracted to the size of a crow-quill, and the coats were thickened. On the posterior wall of the stomach there was a dense cicatrix, of the size of half a crown, and from this point fibrous bands ramified in various directions. The duodenum and other parts of the intestinal canal were healthy. (Med. Times and Gazette, Nov. 26, 1853, p. 554.) A case occurred to Mr. Procter, in May, 1852, in which a woman gave to her infant, four weeks old, a teaspoonful of hartshorn of the strength of about nine per cent. The child became more and more depressed, and died thirty-six hours after taking the liquid. There was no vomiting or purging, and the mouth and throat presented no excoriation; there was, however, slightly increased redness of the lining membrane. An examination after death was not made.

The salts of ammonia are not often used by persons who are intent upon suicide or murder, but there is one instance on record in which a man was tried for the murder of a child by administering to it spirits of hartshorn. (Regina v. Haydon, Somerset Spring Assizes, 1845.) Of the action of the other compounds of ammonia on man, nothing is known.

Chemical analysis.—The three alkalies,—potash, soda, and ammonia, are known from the solutions of the alkaline earths by the fact, that they are not precipitated by a solution of carbonate of potash. They all three possess a powerful alkaline reaction on test paper, which, in the case of ammonia, is easily dissipated by heat. *Ammonia* is immediately known from potash and soda by its odor and volatility. If the solution in water be very dilute, the odor may be scarcely perceptible. The alkali may then be discovered, provided we have first assured ourselves, by evaporating to dryness a portion of the liquid, that other alkalies and alkaline salts are absent. By adding to the solution a mixture of arsenious acid and nitrate of silver, the well-known yellow precipitate of arsenite of silver will be instantly produced. The same result takes place when a carbonate (even bicarbonate of lime) is present; but if a carbonate or other salt existed in the liquid, it would be left on evaporation. In addition to these characters, ammonia redissolves the brown oxide of silver, which it precipitates from the nitrate, while potash and soda do not. (For the detection of ammonia in *organic mixtures*, see ante, p. 298.) The *carbonate of ammonia* may be known from other salts by its alkaline reaction, its odor, and its entire volatility as a solid: from pure ammonia—1. By its solution effervescing on being added to an acid; 2. By its yielding an abundant white precipitate with a solution of chloride of calcium: from the carbonates of potash and soda, among other properties—1. By its giving no precipitate with a solution of the sulphate of magnesia; 2. By the rich violet-blue solution which it forms when added in excess to a weak solution of sulphate of copper; 3. By its odor and volatility.

[*Spirit of hartshorn* is a name applied either to ammonia, carbonate of ammonia, or a mixture of the two.]

CHAPTER XX.

POISONING BY THE ALKALINE SALTS—BINOXALATE OF POTASH (SALT OF SORREL)—BITARTRATE OF POTASH (CREAM OF TARTAR)—NITRATE OF POTASH—SULPHATE OF POTASH—SULPHATE OF ALUMINA AND POTASH (ALUM).

SOME of the alkaline salts have been found to exert an irritant action on the system. The pure alkalies and their carbonates have a corrosive (chemical) action when concentrated, but they operate as irritants when diluted. The salts about to be described are not very energetic poisons, and, with one or two exceptions, require to be given in large doses in order to produce noxious effects.

BINOXALATE OF POTASH (SALT OF SORREL).

The poisonous effects of this salt entirely depend on the oxalic acid which it contains. It is much used for the purpose of bleaching straw and removing ink-stains—being sometimes sold under the name of essential salt of lemons. The smallest quantity retailed to the public is a quarter of an ounce, and for this three half-pence is charged. Its poisonous properties are not commonly known, or no doubt it would be frequently substituted for oxalic acid. Out of three cases of poisoning by this substance two proved fatal, while in the third the patient recovered.

Symptoms and appearances.—In the case of recovery, a young lady, æt. 20, swallowed an ounce of the salt dissolved in warm water. She was not seen by any one for an hour and a half; she was then found on the floor, faint and exhausted, having previously vomited considerably. There was great depression, the skin cold and clammy, the pulse feeble, and there was a scalding sensation in the throat and stomach. There was also continued shivering. Proper medical treatment was adopted, and she recovered in two days,—still suffering from debility and great irritation of the stomach. During the state of depression, it was remarked that the whites of the eyes were much reddened, and the pupils dilated. There was great dimness of vision. (Med. Gaz. xxvii, 480.) In another of the cases,—a lady recently confined took by mistake half an ounce of the binoxalate, instead of cream of tartar. She had scarcely swallowed the draught, when she was seized with violent pain in the abdomen and convulsions; she died in *eight minutes*. On inspection, the mucous membrane of the stomach and small intestines was found inflamed. (Ann. d'Hyg. Avril, 1842.) In the third case, a teaspoonful of this salt was taken for three successive mornings;—it produced severe vomiting; and about an hour after the third dose, the patient expired. We learn from these cases, that this salt is poisonous, destroying life almost as rapidly as oxalic acid itself; and that in the symptoms which it produces, it closely resembles that poison. In the second case, it destroyed life in so short a time as *eight minutes;* but probably the fatal effects were in this instance accelerated by the debilitated state of the person who took it.

Chemical analysis.—The *solid* salt is commonly seen in the form of a crystalline powder, or loosely crystallized in masses. It is not very soluble in cold water, easily taken up on boiling, but reprecipitated in great part on cooling. Its solution might be readily mistaken for oxalic acid; for, 1st, it has an acid reaction; and 2d, it is precipitated by nitrate of silver and sulphate of lime, like oxalic acid; but with respect to the latter test, the precipitation, although more slowly produced, is much more copious. It is known from oxalic acid—1. By its crystalline form, which, as seen in a few drops evaporated on glass, is plumose;

and, 2. By heating a portion on platina-foil. While oxalic acid is volatile, the binoxalate leaves an ash, which, when sufficiently calcined, is alkaline, and it may be proved to contain potash by its being dissolved, with effervescence, by diluted nitric acid, and forming nitrate of potash. There is one vegetable salt for which it has been fatally mistaken, namely, bitartrate of potash, or cream of tartar. This latter is known by its diluted solution not being precipitated by the sulphate or any salt of lime; and by its being rendered only milky or turbid on the addition of nitrate of silver. Lime-water furnishes a ready means of distinguishing these two salts. It precipitates both of them white, but the precipitate from the bitartrate of potash is redissolved on adding to it a small quantity of a solution of tartaric acid, while that from the binoxalate is not redissolved. It may be as well to mention another simple means of distinction,—the color of ink is immediately discharged by warming it with a few grains of binoxalate, but it is unaffected by the bitartrate of potash.

In organic mixtures the process is the same as for oxalic acid itself. (See p. 288.) Although the salt is a natural constituent of sorrel, this vegetable is rarely used as an article of food in England, and then only in small quantity. According to Mitscherlich, the proportion of binoxalate is only 0·75 per cent. of the weight of the fresh plant, or 3·75 per cent. of the juice; hence, one ounce of fresh sorrel will yield but little more than three grains of the salt. The objection suggested by Orfila, that the salt found in a dead stomach might be due to the presence of sorrel taken in soup, is therefore inadmissible, except where the salt is found in traces, and no symptoms of poisoning have existed.

BITARTRATE OF POTASH. (CREAM OF TARTAR. ARGOL.)

Symptoms and appearances.—This salt has proved fatal in at least one instance, although it is not commonly regarded as a poison. The case occurred in this metropolis, in 1837, and is reported by Mr. Tyson. A man, aged 37, took four or five tablespoonfuls of cream of tartar. He was seized with violent vomiting and purging. There was pain in the abdomen, thirst, feeble pulse, and the thighs and legs appeared paralyzed. The fluid vomited was of a dark-green color, and the motions had the color of coffee-grounds. Death took place in about forty-eight hours. On inspection, the mucous membrane of the stomach and duodenum was found highly inflamed, the cardiac portion of the former being of a deep red color, with some spots of black extravasation. The stomach contained a thick brown fluid, colored by bile. The whole of the intestinal canal was more or less inflamed. According to Wöhler, this salt passes off by the urine, under the form of carbonate of potash, the secretion being alkaline. Belloc relates a case of alleged poisoning by Rochelle salt,—the compound tartrate of potash and soda. The circumstances, however, render his statement somewhat doubtful. (Cours de Méd. Lég. 189.)

Chemical analysis.—Cream of tartar is commonly seen in the form of a white powder. It is sparingly soluble in cold water, producing a slightly acid solution. If the *powder* be heated on platina-foil, it is converted to carbon and carbonate of potash; the latter is dissolved with effervescence in acids, and the nature of the alkali is thereby determined. The decomposition of the powder by heat, indicates that it is a vegetable acid salt. On adding the aqueous solution of cream of tartar to lime-water, a white precipitate is formed, which disappears on adding a further quantity. This proves that the acid is the tartaric. It is known from the simple tartrate of potash by its acidity, and by the fact that it is not precipitated by a salt of lime, while the tartrate is precipitated as a tartrate of lime. Chloride of platina gives no precipitate in a cold saturated solution. *Organic mixtures.*—The salt being comparatively insoluble in water, may be found as a sediment at the bottom of the liquid. If dissolved, the liquid may be concentrated and alcohol added; cream of tartar is insoluble in alcohol, and organic matter may be thus separated from it. If the organic liquid be strongly colored, purified animal charcoal should be used to decolorize it. In detecting this substance in the stomach, it is proper to bear in mind, that it is a natural constituent of the potato.

NITRATE OF POTASH. NITRE. SALTPETRE.

This well-known salt is largely employed in the arts. It is an irritant, but acts only as such when taken in a large dose. It has destroyed life on several occasions. Its effects are, however, somewhat uncertain. An ounce, and even two ounces, have been taken without causing very alarming symptoms. (Ed. M. and S. J. xiv, 34.) Dr. Bennett states that M. Gendrin was in the habit of giving it in doses varying from six to twelve, or sixteen drachms in the twenty-four hours without any dangerous symptoms resulting. (Med.-Chir. Review, April, 1844, 549.) M. Mozade has given it with benefit in cases of dropsy in from three to five drachm doses. (L'Union Médicale, 3 Juin, 1847, 274.) According to Tourtelle, no injury has followed, even in cases where it was given in doses of an ounce. (Galtier, Toxicologie, i, 268.) Tartra denied that it had poisonous properties even in a very large dose (op. cit. 135); but cases have occurred which now leave no doubt upon the subject.

Symptoms and appearances.—In one instance, quoted by Orfila, an ounce of nitre was taken by a lady in mistake for other salts. In a quarter of an hour, she suffered from nausea, vomiting, and purging; and the muscles of the face were convulsed. The pulse was weak, the respiration laborious, and the limbs were cold; but there was a sense of burning heat and severe pain at the pit of the stomach. She died in *three hours* after taking the dose. On inspection, the stomach was found highly inflamed, and the membrane detached in various parts. Near the pylorus, the inflammation had a gangrenous character. A large quantity of liquid colored by blood was found in the stomach. (i, 283.) In another case, which proved fatal in sixty hours, where an ounce and a half of nitre had been taken, a small perforation was found in the stomach. (Ib.) I am indebted to Dr. Geoghegan, of Dublin, for the following case: A man took from an ounce to an ounce and a half of nitre by mistake for salts. Severe pain in the abdomen followed, with violent vomiting, but no purging so far as could be ascertained. He died in about *two hours* after taking the salt. On examining the body, a bloody mucus was found in the stomach,—the lining membrane was of a brownish-red color, generally inflamed, and in parts detached from the coat beneath. None of the poison could be detected in the stomach; but its nature was clearly established from the analysis of a portion left in the vessel which had contained the draught. Two men swallowed, each one ounce of nitre by mistake for Glauber's salt. They almost immediately experienced a sense of coldness in the course of the spine, trembling in the limbs, with violent vomiting and purging. The evacuations were bloody. They recovered in the course of a few days. (Casper's Wochenschrift, xviii, 1841.) A case is reported in the same journal, in which one ounce of nitre killed a man in thirty-six hours. In another case an old man, æt. 60, lost his life from an overdose of nitre which he had taken as a medicine. The dose amounted to about ten drachms: it caused profuse purging, and death in about five hours. Death was referred to inflammation of the mucous membrane of the stomach and bowels, owing to the irritant action of the nitre. A female, æt. 28, swallowed in two doses, taken on two days, about an ounce of nitrate of potash. After the second dose, she was attacked with severe burning pain in the stomach, and violent vomiting, followed by collapse. There was no purging, and the secretion of urine was arrested. The girl recovered in a few days. (Pharm. Journal, February, 1846, 356.) Mr. Gillard met with a case in which a man recovered in four days after having swallowed two ounces of nitrate of potash by mistake for Epsom salts. In about five minutes after taking the nitre, he felt a burning pain in his stomach, and this was immediately followed by sickness. Free vomiting was excited by mustard; this probably led to his recovery. (Prov. Med. Journ. August 19, 1846, 382.)

Other cases of recovery after large doses are reported. A man, æt. 80, who had taken nitre medicinally,—half an ounce, in divided doses in the twenty-four hours,—took twelve doses at once. He immediately became insensible, and his face was pale and collapsed as in cholera. The skin was of a marble coldness, the pulse slow, small, and feeble, as was also the respiration. An ipecacuanha emetic restored the power of speech, and he complained of severe burning pains in the throat and abdomen: blood was passed in the evacuations and urine. There was trembling, with slight convulsions, which, together with hallucinations of the senses, and a partial paralysis of muscular action, indicated an operation of the poisonous substance on the nervous system. Free local bleeding, anodyne poultices, and abundant drinks and enemata of milk and linseed-tea, were directed against the inflammatory action, while the great depression and other nervous symptoms were met with camphor and ether. Although dangerous symptoms were thus dissipated, the patient long suffered from derangement of the digestive and urinary organs, and complained of a peculiar feeling of coldness in the hands and feet and down the back. (Berlin Med. Zeitung, 1855, No. 49. Med. Times and Gazette, August 30, 1856.) A woman swallowed an ounce of nitre in two teacupfuls of water. She immediately vomited. When seen the following day, she was sitting with her legs drawn up, the surface of the body pale, but warm and moist. There was constant vomiting. The abdomen was swollen, but soft and tender to the touch. She was unable to move for a week, and then suffered from severe purging with griping:

the evacuations were bloody. She recovered, but remained weak for a long period. (Med. Times and Gazette, Nov. 7, 1857, p. 484.) These facts show that the effects of nitre, although serious, are very uncertain in their character and duration.

Poisoning by nitre has been hitherto the result of accident. It is not taken for the purpose of suicide,—the popular opinion being, that it is not poisonous; although the above cases show that it destroys life with greater rapidity than is commonly observed in the action of arsenic and corrosive sublimate. It is not likely to be employed by a murderer, since a dose sufficient to kill, could not be unconsciously taken.

Treatment.—There is no antidote known. Mucilaginous drinks should be given: vomiting should be freely promoted, and the stomach-pump used for the removal of the salt.

Chemical analysis.—(See Nitric Acid, ante, p. 260). From the researches of Wöhler, it would appear that nitre is *absorbed* and eliminated in the urine. He detected it in the urine of a horse four hours after he had given to the animal five ounces of nitre. M. Reynard also detected it in the urine of persons to whom it had been given medicinally. His plan consisted in throwing down the sulphates and phosphates of the urine by a solution of baryta; filtering, evaporating to dryness, and treating the residue with pure alcohol in order to dissolve out urea and other substances soluble in this menstruum. The nitre was then procured by digestion in water, evaporation, and crystallization. (Galtier, Toxicologie, i, 262.) Orfila states that he has detected nitre in the liver, spleen, kidneys, and urine of animals poisoned by it. (Ann. d'Hyg. 1842, ii, 434.)

SULPHATE OF POTASH.

This salt was formerly called *Sal de Duobus* or *Sal de Polychrest.* It has been regarded as inert, but of late years it has given rise to some important medico-legal investigations.

Symptoms and appearances.—A lady, about a week after her delivery, took, by the prescription of her medical attendant, about ten drachms of the sulphate of potash in divided doses, as a laxative. After the first dose, she was seized with severe pain in the stomach, nausea, vomiting, purging, and cramps in the limbs. These symptoms were aggravated after each dose, and she died in *two hours.* It was supposed that some poison had been given by mistake; but that was not the case, and the question was, whether her death was or was not caused by the sulphate of potash. On an inspection of the body, the mucous membrane of the stomach and intestines was found pale, except the valvulæ conniventes (folds), which were reddened. The stomach contained a large quantity of reddish-colored liquid, which, on analysis, was found to contain only sulphate of potash, and no trace of any common irritant poison. The examiners referred death to sulphate of potash taken in an unusually large dose, whereby it had acted as an irritant poison on a person whose constitution was already much debilitated. (Ann. d'Hyg. Avril, 1842.)

The question whether this is to be regarded as an irritant poisonous salt or not, was much debated among members of the profession, in reference to a case which was tried at the Central Criminal Court in October, 1843. (The Queen v. Haynes.) The accused had given to the deceased, the night before her death, two ounces of sulphate of potash, dissolved in water; and it was alleged that a fortnight previously to this, she had taken in divided doses as much as a quarter of a pound of the salt. The woman thought that she was pregnant, but this was disproved by an examination of the body; and it was charged that the prisoner had given her the salt with the intention of causing a miscarriage. After the last dose, she was seized with sickness, and died within a very short time. The stomach was found empty, but highly inflamed; and there was blood effused on the brain. One medical witness referred death to the action of this salt as an irritant poison; the other to apoplexy, as an indirect result of the violent vomiting caused by it. The prisoner was acquitted of the charge of murder, but subsequently found guilty of administering the sulphate with intent to procure abortion. Both of the witnesses admitted that, in small doses, the salt was innocent; but that in the dose of two ounces it would produce dangerous effects. A portion of the sulphate in this case was examined by Mr. Brande, as it was suspected that some poisonous substance might have become accidentally mixed with it; but it was found to be pure. It is not improbable, from the symptoms and the inflamed state of the stomach, that the salt acted here as an irritant poison; and the fact of its being an innocent medicine in small doses appears to be no sound objection to this view; for the same circumstance is observed with respect to numerous substances, the poisonous or noxious properties of which cannot admit of dispute (ante, p. 18.)

A case, somewhat similar in its details, was the subject of a trial at the Central Criminal Court in October, 1856. (Reg. v. Gaylor.) A married woman, the wife of the prisoner, under the belief that she was pregnant, took a large quantity of this salt, the prisoner having purchased two ounces, and handed it to her. It was taken with the design of procuring abortion, but it caused the death of the woman under symptoms of severe irritation of the stomach and bowels. The deceased was not seen by a medical man while living, but she suffered from severe pain, vomiting, and purging: the vomited matter had a bilious color. On inspection, the stomach and the upper portion of the smaller intestines were of a deep purple color, as if from the action of some irritating substances. The stomach, when opened, showed marks of irritation, and its mucous coat was much congested. In this organ there was a spoonful of thick, slimy fluid, which contained a quantity of sulphate of potash. The intestines contained twelve ounces of a thick, white fluid, highly charged with mucus, and this, when analyzed, yielded sulphate of potash.

There was no doubt that death had been caused by an overdose of this substance: but there was the usual legal doubt whether the prisoner had committed any crime in handing it to the deceased. According to Mr. Mowbray (Medical Gazette, xxxiii, p. 54), sulphate of potash is a salt much employed in France as a popular abortive. He quotes several instances in which, in large doses, it produced severe symptoms, resembling those of irritant poisoning, and even death. In one case, two drachms acted powerfully: and in another, that fell under his own observation, four drachms of the salt, administered to a lady after her confinement, had all the effects of an irritant poison. The above cases are the only instances in which, I believe, it is publicly known to have proved fatal in England; and they show that substances, commonly regarded as innocent, may give rise to important questions in toxicology.

There is no doubt that the most simple purgative salts may, under certain circumstances, and when given in large doses, destroy life. A case has been already related (ante, p. 19), in which sulphate of magnesia caused death, and gave rise to a criminal charge in this country. It is said that sulphate of potash has in some cases caused vomiting and other serious symptoms, from its containing as impurity sulphate of zinc. This would be easily discovered by adding the ferrocyanide of potassium, which gives a white precipitate with a salt of zinc.

Chemical analysis.—Sulphate of potash is easily identified. It is a dry, hard salt, soluble in water, forming a neutral solution. This solution, if sufficiently concentrated, is precipitated both by tartaric acid and bichloride of platina, whereby potash is indicated; and the presence of sulphuric acid is known by the action of a salt of baryta. *Organic liquids.*—This salt being insoluble in alcohol, may have the organic matter removed from it by treating the liquid containing it (previously concentrated) with alcohol: or the substance containing the salt may be evaporated to dryness, and incinerated, when the undecomposed sulphate may be obtained by lixiviating the calcined residue with distilled water. The sulphate of potash exists naturally in some animal fluids, but only in minute traces.

ALUM. SULPHATE OF ALUMINA AND POTASH.

This substance is very commonly known; but it does not appear to have given rise to many accidents, at least in this country. One case of death from alum, appears in the Registration Report for 1838–9. A singular case occurred in Paris, in 1828, in which the alleged noxious properties of alum were brought into question. A lady swallowed a quantity of calcined alum dissolved in warm water, which had been supplied to her by mistake for powdered gum. The quantity taken was less than half an ounce. She immediately complained of a burning pain in the mouth, throat, and stomach. She afterwards suffered from thirst, violent vomiting, and general disturbance of the system, from which she recovered in the course of two or three days. These effects were referred to the alum, and the party who supplied it by mistake was condemned to a severe punishment. On the case being carried to an appeal, Orfila contended that alum was not a poison; although he admitted that in the calcined state it was a caustic, and in order to establish his opinion of its inertness, he offered to swallow half an ounce on the spot! He referred the symptoms under which the party labored to some other cause: but, on being further questioned, he admitted that a solution of calcined alum was likely to produce more serious effects than common gum, which the party should have taken. The punishment was mitigated. (Ann. d'Hyg. 1829, i, 234.) Orfila subsequently ascertained by experiment that alum in a large dose operated fatally on animals, destroying life in the course of a few hours! He detected the salt in these cases in the substance of the stomach, liver, spleen, and in the urine. (Ann. d'Hyg. 1842, ii, 433.) The reader will find a singular case of supposed poisoning by alum in the Ann. d'Hyg. 1832, ii, 180.

The *symptoms* produced by alum in a large dose, are frothing at the mouth, vomiting (the vomited matters containing alum), purging, depression, weakness of the limbs, and the principal *appearance* is a reddish-brown color of the mucous membrane of the stomach, which may be found softened or disorganized, either wholly or in patches. We cannot, therefore, refuse to admit the fact of this substance acting as an irritant, on the principle on which we admit the irritant properties of salts of a far more innocent character. It is, however, proper to observe, that alum, given in large doses to animals, does not appear to affect them seriously unless the gullet be tied; *three drachms*, dissolved in six ounces of liquid, have been taken at a dose without any inconvenience resulting.

Treatment.—The promotion of vomiting and the free administration of hydrate of magnesia, or a weak solution of carbonate of ammonia at intervals.

Chemical analysis.—Common alum possesses a peculiarly acid and astringent taste. It is easily dissolved by water, forming an acid solution, which crystallizes on evaporation in regular octahedra. Its solution is not affected by ferrocyanide of potassium or sulphuretted hydrogen gas, whereby it is known from the true metallic saline solutions. The sulphuric acid may be detected by a salt of baryta. On adding potash, a white precipitate of alumina falls down, which is redissolved by the addition of a larger quantity of the alkali. By this last character, it is known from the alkaline earths, which are precipitated from their solutions by potash, but the precipitates are not redissolved. On adding carbonate of ammonia, alumina falls down. This may be separated by filtration, and on evaporating the liquid portion, and incinerating the saline residue, there will be found sulphate of potash. Calcined alum is a white uncrystalline substance, only partially soluble in water. About one-sixth is left as a residuary white powder, easily soluble in a mineral acid, and yielding common alum by crystallization. The quantity dissolved by boiling water is, however, sufficient to allow its nature to be determined. From *organic liquids* it may be obtained by evaporation and incineration.

CHAPTER XXI.

METALLOIDS—PHOSPHORUS—SYMPTOMS AND APPEARANCES—CHRONIC POISONING BY THE VAPOR—FATAL DOSE—CHEMICAL ANALYSIS—PHOSPHORUS-PASTE—RED OR AMORPHOUS PHOSPHORUS.

Phosphorus.—Cases of poisoning by phosphorus appear to have been much more numerous in France than in England. M. Chevallier has collected seventy-four cases of poisoning by this substance, and in forty-two of these, the phosphorus was procured from lucifer matches. Among the cases, twenty-five were the result of accident, twenty-eight involved a criminal charge, and twenty-one were the result of suicide. (Ann. d'Hygiène, 1857, vol. ii, p. 226.) The cases of poisoning by phosphorus in this country are not very numerous. They are chiefly referable to accident or suicide. One trial for the criminal administration of phosphorus took place at the Bodmin Autumn Assizes, 1857, in which there was no doubt that death had been caused by phosphorus-paste.

SYMPTOMS.—Phosphorus acts as an irritant poison, but its operation is attended with some uncertainty, according to the state in which it is taken. The symptoms are slow in appearing; it is only after some hours, and sometimes even one or two days, that signs of irritation with convulsions and spasms appear; but when these once come on, the case proceeds rapidly to a fatal termination. In the first instance, the patient experiences a disagreeable taste resembling garlic, which is peculiar to this poison. An alliaceous or garlic odor may be perceived in the breath. There is an acrid burning sensation in the throat, with intense thirst, severe pain and heat with a pricking sensation in the stomach followed by distension of the abdomen; and nausea and vomiting continuing until death. The vomited matters are of a dark green or black color; they have the odor of garlic, white vapors may be seen to proceed from them, and in the dark they may even appear phosphorescent. Purging is among the symptoms, and the motions have been observed to be luminous in the dark. The pulse is small, frequent, and scarcely perceptible. There is great prostration of strength, coldness of the skin, and other symptoms of collapse. The patient may die quietly, or be convulsed before death. The following cases will illustrate the mode in which this poison acts.

A girl, between two and three years of age, had been caught in the act of sucking and swallowing the heads of lucifer matches. Two days afterwards she appeared unwell,—there was some feverish excitement, but no active symptoms. The bowels were open, but the child did not suffer from pain, vomiting, or purging. Five hours after she was first seen, she became violently convulsed, and she died in three hours. On inspection, a quantity of mucus mixed with blood, of a coffee-ground color, was found in the stomach. The mucous membrane was very red throughout, and for the space of about two inches it had a florid-red color, and was covered with mucus. There were no fewer than ten invaginations in the small intestines (intussusception, ante, p. 126), many of which included from two to three inches of intestine, which was inflamed at the invaginated parts. There was no appearance of strangulation, and the bowels were empty. The medical opinion given at the inquest was, that phosphorus, in a finely-divided state, was the cause of death, and a verdict was returned accordingly. (Lancet, Dec. 1843.) A female committed suicide by dissolving in vinegar the phosphorus from the ends of some lucifer matches. She swallowed this mixture, and after undergoing the most severe suffering for eight days, she died laboring under symptoms resembling those of hydrophobia. (Journ. de Chim. Méd. 1846, 668.)

A boy, æt. 10, took medicinally phosphorus in pills and in an oleaginous mixture for nearly four weeks. He was found lying in a state of stupor, quite insensible, laboring under strong convulsions, hurried breathing, and a small pulse. He died some hours afterwards. The principal appearances were congestion of the brain, a bright vermilion color of the anterior surface of the stomach externally, with softening of the mucous membrane within,—and the marks of violent irritation and inflammation of the muscular coat of the large intestines. The quantity of phosphorus taken is not stated; it was given in divided doses, and none had been taken for ten days previously to death; nevertheless death was ascribed to the long-continued use of the substance. The stomach contained two ounces of a dark-brown liquid, and a large quantity of mucus. (Lancet, Sept. 14, 1844.) A case is reported by Dr. Graff, in which a young woman swallowed the phosphorus obtained from about three hundred matches,—equal to rather less than *five grains* of pure phosphorus, —and recovered from the effects. The symptoms do not appear to have been very severe,—a fact ascribed by the reporter to the phosphorus having been in an intimate and probably insoluble state of combination with other substances in the matches. (Henke, Zeitschrift, 1842, ii, 288.) Phosphorus in small doses is said to produce strong aphrodisiac effects. This view is borne out by the facts collected by Dr. Hartcop. (See Casper's Wochenschrift, 21 Feb. 1846, p. 115.)

Phosphorus vapor. Chronic poisoning.—Chronic poisoning by phosphorus is accompanied by nauseous eructations, frequent vomiting, sense of heat in the stomach, purging, straining, pains in the joints, wasting, hectic fever, and disease of the stomach, under which the patient slowly sinks. Some interest is attached to the chronic form of poisoning by phosphorus from the researches of Dr. Strohl and others on the effects of the *vapor* upon individuals engaged in the manufacture of phosphorus or lucifer matches. It has been remarked that persons thus engaged have suffered from necrosis of the jaw, carious teeth, and abscesses. There has been also marked irritation of the respiratory organs, and bronchitis has frequently shown itself among them. These effects have been attributed to the respiration of the vapors of phosphorus, which are supposed, by becoming acidified, to act chemically upon the bones. A good summary of the facts, connected with this kind of poisoning, by Dr. Beck, will be found in the American Journal of Medical Sciences, for Oct. 1846, 525. (See also Annales d'Hygiène, 1856, ii, p. 5; and 1857, i, p. 431.) A case in which pneumonia was considered to have been induced by phosphorus vapor, is reported in the Med. Gaz. (vol. xxxix, p. 210), and another well-marked instance of the serious local and constitutional effects of the acid vapors has been published by Mr. Wright. (Med. Times, Dec. 19, 1846, 224.) According to M. Dupasquier, phosphorus in vapor has no specific poisonous action: it merely irritates the lining membrane of the bronchial tubes, and this effect is soon lost by habit. When other and more dangerous symptoms supervene, he thinks they should be ascribed to the accidental presence of arsenic in the phosphorus. (Journal de Pharmacie, Oct. 1846, 284; also, Gaz. Méd. Dec. 5, 1846, 946.) This view of M. Dupasquier is not borne out by experience. Numerous facts are now placed on record which show that the vapor of phosphorus produces most injurious effects to health.

APPEARANCES.—The appearances produced by this poison have been already described in two of the cases above given. We may be prepared to find marks of irritation and inflammation in the stomach and intestines. The stomach has been observed to be much contracted. The mucous membrane will be found inflamed and probably softened. Inflammation of the stomach and bowels proceeding to gangrene is a common result of the action of phosphorus. M. Worbe found the stomach perforated in three places in a dog which had been poisoned by a solution of phosphorus in oil. A man, æt. 50, took a quantity of phosphorus-paste used for destroying vermin. He was seen in his usual health at twelve o'clock P.M., and was found dead in a field the following morning. On inspection, it was observed that there was great muscular rigidity. The membranes of the brain were much congested, and there was serous effusion between the arachnoid and pia mater. The substance of the brain was also congested. The heart was flaccid and nearly empty. The mucous membrane of the stomach, gullet, and small intestines, was very red, and there were patches in which the membrane was destroyed. On opening the stomach a white smoke escaped, accompanied by a strong smell of garlic. It contained a tablespoonful of viscid greenish matter, from which particles of phosphorus with some Prussian blue (used as a coloring for the poison), subsided on standing. (Dr. Bingley, Lancet, June 13, 1857, p. 600.) Mr. Herapath states that in a case which he examined (ante, p. 179), he found, besides inflammation of the stomach, the mucous membrane raised in small bladders or vesications. This was no doubt a change produced by putrefaction, as the body was not examined until twenty-three days after death. Such a blistered appearance is frequently seen in cadaveric inspections, and has not been observed in recent cases of poisoning by phosphorus. Schuchardt describes, among the appearances, fluidity of the blood, which is of a dark color, and does not become red on exposure to the air. Ecchymoses are sometimes found on the skin and on the surface of various organs. (Brit. and For. Med. Rev. 1857, vol. xix, p. 506. Journal de Chimie Médicale, 1857, p. 84.) The viscera and even the flesh of animals recently poisoned by phosphorus, have the odor of garlic, and appear luminous in the dark. (Galtier, Toxicologie, i, 184.) In the case of a woman who died while taking phosphorus medicinally, it was remarked that the whole of the viscera of the body were luminous; thus indicating the extensive diffusion of the poison. (Casper's Wochenschrift, 21 and 28 Feb. 1846, 115, 135.) For a further account of the appearances, see Chemist, Jan. 1856, p. 244.

Absorption.—That this poison is absorbed and diffused through the body is established by the luminosity of the viscera, which has been frequently observed. Vauquelin, after having exposed himself to the vapor of phosphorus, observed that the urine which he passed was phosphorescent, and M. Chevallier states, on the authority of a phosphorus-manufacturer, that on many occasions the men who were employed in his establishment, and who were in the habit of breathing phosphorous vapor, passed phosphorescent urine. (Annales d'Hygiene, 1857, vol. ii, p. 214.) It is not improbable that this substance may be eliminated by the lungs, and that the breath of persons poisoned by phosphorus may be luminous in the dark.

Fatal dose.—That phosphorus is a powerful poison, is proved by two cases quoted by Dr. Christison. In one, death was caused by a grain and a half in twelve days; in the other, by two grains in about eight days. It is supposed to operate as a poison only by becoming converted to phosphorous and phosphoric acids; but although this conversion takes place, it is probable that it passes directly into the blood, since the urine passed during life has been observed to be luminous. The production of these acids may account for the erosions met with in the stomach and bowels. A person has been killed by a quantity of phosphorus (case supra) equal to less than three and a half grains of phosphoric acid, while fifty grains of phosphoric acid have been given to a rabbit without effect! The fatal dose is liable to vary according to many circumstances. Galtier states that it is comprised between three-quarters of a grain and two grains, and that even a third of a grain has destroyed life; while persons have recovered, as in one instance referred to, from a dose of *five* grains (p. 313). In a case reported by M. Worbe, and quoted by Orfila, the ascertained fatal dose was less than *a grain and a half*. The subject was a man aged 27. The phosphorus was melted in hot water, and thus swallowed. Three days previously the man had taken less than half a grain (three centigrammes) without any ill effects. The patient suffered from all the symptoms of irritant poisoning, and died in twelve days. It is worthy of remark, however, that no striking symptoms showed themselves for several hours. (Toxicologie, i, 55.)

Dr. Hartcop mentions that an apothecary took by way of experiment one grain,—the next day two grains, and the third day three grains of phosphorus mixed with sugar. He was then seized with inflammation of the stomach and bowels, and died in spite of every attempt to save him. (Casper's Wochenschrift, 1846, p. 117.) M. Chevallier refers to a case in which a dose of 2·3 grains proved fatal, and to two other cases in each of which a dose of 4·6 grains destroyed life. The same writer quotes, on the authority of Löbenstein Löbel, of Jena, the case of a lunatic who died from a dose of one-eighth of a grain. (Ann. d'Hyg. 1857, vol. i, p. 422.) Excepting this, the smallest fatal dose which I have met with, is in a case quoted by Galtier. A woman, æt. 52, took in divided doses, in four days, about six centigrammes, or less than *one grain*, of phosphorus dissolved. The largest dose taken at once, *i. e.* on the fourth day, is stated to have been three centigrammes (0·462 grain), or less than half a grain. Symptoms of pain and irritation appeared, and the patient died in three days. The gullet, stomach, and small intestines were found much inflamed. (Toxicologie, i, 87.) One-quarter of a grain dissolved in oil, has been known to produce burning heat in the abdomen, vomiting, and purging; and from one-quarter to one-eighth of a grain dissolved in oil, has produced severe symptoms of inflammation of the stomach. (Böcker, Vergiftungen, 1857, p. 127.) The physical state of the phosphorus is an important element in considering the fatal dose. Phosphorus can only act by its surface, and thus a stick of it might be swallowed and after a time rejected by vomiting, without doing the injury which would be occasioned by a much smaller quantity dissolved. M. Tilloy saw a cat swallow a stick weighing six grains, and reject it without manifesting any injurious symptoms. (Ann. d'Hyg. 1857, vol. i, p. 432.) When the phosphorus is dissolved in any liquid or when it is finely divided, as in phosphorus-paste or in lucifer matches, its action is then more powerful, as it is in a state well fitted for absorption.

PERIOD AT WHICH DEATH TAKES PLACE.—The earliest period is in a case related by Orfila, in which a young man in weak health took a dose of phosphorus (the quantity unknown) and died in *four* hours. The same author mentions a case in which death did not take place until after the lapse of seventeen days. In general, several days elapse before a fatal result takes place, and during this time the patient undergoes much suffering. This was observed in a young female who swallowed a quantity of phosphorus-paste intended for poisoning rats. She did not die until the fifth day. (Journal de Chimie Méd. 1845, 580.)

TREATMENT.—This may consist in the administration of emetics and of albuminous or mucilaginous drinks holding hydrate of magnesia suspended, as well as in the free use of emetics and purgatives. When the symptoms have once manifested themselves, it is difficult to arrest their progress, and there is no known antidote to the poison. The exhibition of oil would be decidedly injurious, as this dissolves and tends to diffuse the poison. The presence of phosphorus in the matters vomited or passed by the bowels is known by the peculiar odor and their luminosity in the dark.

CHEMICAL ANALYSIS.

Phosphorus is a solid of waxy consistency, having a peculiar odor and taste resembling garlic. It is this odor and taste which prevent it from being criminally employed as a poison, and leads to its detection in articles of food. It evolves a white vapor in daylight, and a faint bluish luminosity in the dark. It melts and takes fire at a temperature a little above 100°, burning with a bright yellow flame and producing thick white acid vapors by combustion. It is not soluble in water, but water in which it has been preserved or washed, acquires poisonous properties by reason of the phosphorous acid formed. (Ann. d'Hyg. 1857, vol. i, p. 423.) It is soluble in alcohol and the oils, but especially in ether, sulphide of carbon, and chloroform.

Organic mixtures.—The smell which phosphorus imparts to organic substances is remarkably characteristic. When it has been taken in a solid form it may be separated as a sediment in fine particles by washing the contents of the stomach in water. If a portion of the organic liquid be exposed in the dark, the particles of phosphorus will be known by their luminosity, as well as their combustion when the surface on which the material is spread is heated. Dr. Neumann met with the following case. A shepherd, after having eaten some beet-root soup, vomited several times, complained of thirst, intense pain in the abdomen, and died after two days' continuous suffering. His dog, which had eaten some of the food, became unwell, and died in two hours. The man lived unhappily with his wife, and, from some suspicion as to the cause of death, the body of the deceased, as well as that of the dog, was ordered to be disinterred and examined. As the bodies had been buried *fourteen* days, and the weather was warm, they were in an advanced state of decomposition. It was impossible to draw any inference of poisoning from the appearances of the viscera. A portion of the soup of which the deceased and his dog had eaten, was procured and submitted to examination. A small quantity was spread on an iron plate, heated to a moderate temperature. Portions immediately burnt with a yellow light and a thick white smoke. In addition to this, the soup had the smell of phosphorus, and was luminous in the dark. (Casper, Wochenschrift, Mai 31, 1854.) In this instance Dr. Neumann converted the phosphorus in the stomach of the man into phosphoric acid by nitric acid, and separated a quantity equivalent to about six grains.

Phosphorus is very soluble in sulphide of carbon, and I find that it may be separated from organic matters by digestion with this liquid. On the spontaneous evaporation of the sulphide, the phosphorus may be procured in small globules or beads. The phosphorus thus obtained sometimes takes fire spontaneously, and burns with its well-known flame.

Another plan for detecting phosphorus in organic liquids consists in its conversion to phosphoric acid by boiling the solid materials, or the residuary particles obtained by washing and decantation, in nitric acid diluted with three parts of water. By evaporating to dryness, and incinerating the dry residue, phosphoric acid may be dissolved out of the ash and tested in the usual way. In some cases, where there is no smell of phosphorus, and no luminosity, this change to phosphoric acid may have already taken place in the body. The evaporation and incineration of the contents with a solution of the residue in water will then suffice. Phosphoric acid, as it is thus procured by heat, gives a *white* precipitate with nitrate of silver, soluble in nitric acid. On adding ammonia and a solution of sulphate of magnesia, an abundant white precipitate is thrown down, which is insoluble in a solution of muriate of ammonia. The tissues may be thus treated for the detection of phosphorus, but in this case due allowance must be made for the presence of normal phosphates in the solids and fluids of the body. (Ann. d'Hyg. 1857, vol. ii, p. 209.)

According to the observations of MM. de Vrij and Van der Burg, the effluvia escaping from a putrefied body do not prevent the luminous appearance of phosphorus. (Journal de Pharmacie, Fevrier, 1857, p. 94.) This fact was noticed in Neumann's case (supra), in which the body had been buried fourteen days, and was highly putrefied. If the luminosity should not be apparent, the addition of a small quantity of sulphuric acid and agitation of the liquid may bring it out. It is well known that ether, alcohol, and oil of turpentine prevent this oxidation of phosphorus by air, and destroy the luminosity.

The process suggested by Mitscherlich for the detection of small quantities of phosphorus, removes this difficulty in so far as alcohol and ether are concerned. Mitscherlich adds to the organic substance a sufficiency of water to make it quite fluid, and a small quantity of sulphuric acid. This mixture is placed in a flask, connected with a long glass condensing-tube, placed vertically and kept cool by a stream of cold water. The tube is fitted into a receiver. The suspected liquid is distilled in the dark, and if a minute trace of phosphorus be present, 1-100,000th part, or, according to De Vrij, 1-2,000,000th, the fact will be made evident by the luminous appearance in the upper part of the tube, at each successive condensation of the vapors. (See Otto, Ausmittelung der Gifte, 1856, p. 78.) If ether or alcohol should be present, the vapors of these liquids are distilled over first. (Die Vergiftungen, Böcker, 1857, p. 83.) In the receiver in which the vapor of the distilled liquid is condensed, phosphorous or phosphoric acid may be discovered by the usual tests.

There can be no doubt of the facility and delicacy with which phosphorus thus admits of detection. A case is elsewhere given (ante, p. 179), in which Mr. Herapath failed to detect any trace of it in a body on the twenty-third day after death: hence, like other poisons, it is liable to disappear from the body.

Phosphorus-paste.—This consists of phosphorus in a finely divided state mixed with a farinaceous paste, and sometimes colored with Prussian blue. The colored sample appears white until exposed to the air, when it changes to blue. The substance has the powerful odor of phosphorus, it fumes in the air, giving off the usual white vapors of phosphorous acid. When spread in a thin layer on a sheet of mica and heated, the particles of phosphorus burn with bright scintillations, and the farinaceous matter is carbonized. The phosphorus may be converted to phosphoric acid by boiling with nitric acid slightly diluted, or it may be dissolved out of the paste by sulphide of carbon. The farinaceous portion of the compound may be known by the addition of iodine and the application of the microscope. This paste is luminous in the dark, giving off a visible phosphorescent vapor. It is colorless when not in contact with air, so that the blue color from Prussian blue may not be seen when the stomach is first opened. This effect of color should be borne in mind. The vomited matter, in poisoning by phosphorus, as well as the contents of the stomach after death, may be blue. If the blue color depends on Prussian blue it will be entirely destroyed by the ammonia of putrefaction. According to one formula, this substance consists of one drachm of phosphorus (finely divided by melting it in rectified spirit), five ounces of flour, and an ounce and a half of brown sugar, made into a paste with a little water. (Pharm. Journal, 1852–3, p. 402.)

Red or Amorphous Phosphorus.—The remarkable body, known under the name of allotropic phosphorus, is not possessed of poisonous properties. This fact, long since announced by Liebig (Letters on Chemistry, 165), has been confirmed by experiments at the Veterinary College of Alfort. (Ann. d'Hyg. 1857, vol. i, p. 432.) Common phosphorus is poisonous in doses varying from one to three grains, while amorphous phosphorus has been given to animals in a dose of thirty-one grains without causing symptoms of poisoning. The amorphous phosphorus, by reason of its being in a fine powder, is in a state more favorable for acting as a poison than common phosphorus, and yet it is inert! M. Bussy, in 1850, and M. de Vrij, in 1851, proved that a dog might take with impunity thirty grains. Orfila and Rigaut have given it to animals in doses amounting to some ounces, over a period of twelve days, without producing any noxious effects. (See Annuaire de Thérapeutique, 1855, p. 103.)

Considering that this body is simply phosphorus in an altered physical state, and that it is again convertible to common phosphorus when heated to a high temperature, we have here the instance of the same substance being poisonous in one molecular condition, and not poisonous in another! Red phosphorus is easily known by heating it in air, when it burns like common phosphorus. It is insoluble in all liquids, and by its insolubility in sulphide of carbon it is distinguished and separated from common phosphorus. It is not luminous in the dark, unless it contains common phosphorus. In any analysis for phosphorus, we must take care to exclude it by employing sulphide of carbon as a solvent for the common or poisonous form of phosphorus. (The reader will find a full account of the comparative effects of the common and red phosphorus by M. Chevallier, in the Annales d'Hygiène, 1856, i, 374.)

CHAPTER XXII.

METALLIC IRRITANTS—THEIR GENERAL CHARACTERS—ARSENIC—PRECAUTIONS RESPECTING THE SALE OF THE POISON—ARSENIOUS ACID—TASTE—SOLUBILITY IN VARIOUS LIQUIDS—SYMPTOMS—CASES OF ACUTE POISONING—CHRONIC POISONING—ANOMALOUS CASES—ALLEGED ACCUMULATIVE PROPERTIES—EFFECTS OF EXTERNAL APPLICATION—APPEARANCES AFTER DEATH—PERIOD REQUIRED FOR INFLAMMATION AND ULCERATION—QUANTITY REQUIRED TO DESTROY LIFE—RECOVERY FROM LARGE DOSES—PERIOD AT WHICH DEATH TAKES PLACE—TREATMENT.

THE metallic irritant poisons do not admit of any easy subdivision, either according to differences in their mode of action or their chemical properties. The chemical differences are often as great among the salts of each metal as among the salts of different metals. They will therefore be here treated chiefly in the order of their practical importance, the compounds of those metals being taken first which most frequently give rise to medico-legal inquiries. We shall, in the first place, pass to the consideration of ARSENIC.

WHITE ARSENIC—ARSENIOUS ACID.

General Remarks.—The term WHITE ARSENIC is commonly applied to the arsenious acid of chemists. Arsenic acid is another compound which is highly poisonous, but has never, so far as I know, been used for the purposes of suicide and murder. YELLOW ARSENIC, or Orpiment, is the sesquisulphuret of chemists. This is also poisonous, apparently because it contains a large proportion of arsenious acid, which has not combined with sulphur. This often amounts to from fifteen to twenty per cent. of its weight. Orpiment has been, on several occasions, criminally used as a poison. White arsenic, or arsenious acid, is, however, that preparation, which chiefly requires the attention of a medical jurist. It is often sold in the country by an ignorant class of dealers, under the name of *mercury*. As a witness may sometimes have to infer *quantity* from the cost, it may be stated, that from half an ounce to one ounce of white arsenic is sold for two pence,—one ounce and a half for three pence—if exceeding this, the charge is at the rate of one shilling per pound. By the 14th Victoria, chap. xiii, sec. 3, the sale of white arsenic in any quantity less than ten pounds is prohibited, unless it be mixed with 1-16th part of its weight of soot, or 1-32d part of its weight of indigo. The vomited matters in a case of poisoning by arsenic may therefore be blue or black,—or the admixture of bile may render them of a deep green color. In a case of arsenical poisoning communicated to me by Dr. Maclagan, the *blue* vomiting at first completely misled those who were called to render assistance. As soot and indigo are both insoluble in water, these substances will be slowly deposited from the vomited matter by subsidence, and the color given by blood or bile may then become perceptible.

A medical witness may be asked the weight of common or familiar measures of arsenic in powder. It may, therefore, be stated that a teaspoonful of finely-powdered white arsenic weighs 150 grains,—a tablespoonful weighs 530 grains,—and a pinch, or the quantity taken up between the finger and thumb of an adult, weighs 17 grains. The weights here given are the results of actual experiments: but they are, of course, liable to vary.

Taste of arsenic.—White arsenic is commonly seen under the form of a white powder, or in opaque masses resembling enamel. It is called an acid from its power of combining with alkalies, but it possesses a very feeble acid reaction when dissolved in water. It is often described as having an *acrid taste*, but this does not appear to be correct; a small quantity of it has certainly no appreciable taste,—a fact, which might be established by direct experiment, and might be inferred from its very sparing solubility. It would appear from numerous cases on record, that it has been unconsciously taken in fatal quantities, in all descriptions of food, without exciting the least sensation on the tongue. Most of those persons who have been criminally or accidentally poisoned by arsenic, have not been aware of any taste in taking the poisoned substance: it has certainly not been perceptible when the poison has been given in wine, milk, beer, and other simple liquids, or many lives would have been saved. In an accident, by which a whole family was poisoned by arsenic mixed in a pudding, for the particulars of which I am indebted to Mr. Tubbs, of Upwell Isle, one person found the taste rather sweetish, rough, and persistent, but experienced, at the same time, a burning sensation in the throat. Another felt his throat so hot, that it was just as if he had swallowed pepper. In another case, which occurred to the same gentleman, there was a better opportunity of judging of the taste of the poison: a teaspoonful (150 grains) of the poison was taken by an adult in the form of dry powder, without any fluid after it: the man described the taste as "coarse and smartish." Others have spoken of it in the state of powder as having a rough or salt taste. M. Flandin considers that arsenious acid has a decidedly hot taste only when it is taken in a very large dose, an experiment, upon the performance of which no toxicologist is likely to venture; and that, in small doses, it is quite insipid. In some cases of poisoning, in which several persons partook of meat on which powdered arsenic had been sprinkled, they stated that the meat had an unusual taste, something like that of a sour apple. (Des Poisons, i, 515.) In a case of arsenical poisoning (March, 1846), for an accurate report of which I am indebted to Mr. J. H. Todd, coroner for Hants, the person swallowed at least a drachm of white arsenic, mixed with half a teacupful of water, before breakfast, and on an empty stomach. These were circumstances highly favorable to the detection of the *taste* of the poison. After he had drunk off the dose, he said it was rather rough to the tongue; and for twenty minutes afterwards he complained that his tongue was very rough. In the case of the Queen v. Maher and Lynam (Kildare Lent Assizes, 1847), some flummery was proved to have been poisoned with arsenic, and the taste of the flummery was complained of. My friend, Dr. Geoghegan, who communicated to me the particulars of this case, says that the taste could not be referred to the after-effects of the poison, and suggests that arsenic, like aconite and some other poisons, may have a taste after a certain interval. The facts above related will enable a medical jurist to form a judgment of the matter. If Orfila has given an exaggerated account of the taste of this poison, it seems clear that, in the state of powder and in large doses, arsenic is, in many cases, capable of producing a decided and persistent impression on the tongue; while it is nevertheless true that when dissolved,—when it is in small quantity, or mixed with common articles of food, the poison may be easily taken by a person quite unconsciously.

Arsenic not a corrosive.—Arsenic is an irritant poison; it has no decided chemical or corrosive action on the animal tissues, and the changes met with in the stomach and bowels of a person poisoned by it, are referable to the effects of inflammation. I have not found that arsenic produces any effect on dead mucous membrane. Nevertheless, it is proper to state, that one instance at least is on record, in which it is alleged to have exerted a corrosive action as a poison. A man, named Soufflard, on being condemned to death, swallowed three drachms of arsenious acid in powder; he vomited almost immediately. When seen shortly afterwards the lower lip was strongly cauterized (*fortement cautérisée*); the mucous membrane was white, fissured, and the slightest touch produced excessive pain. The tongue was swollen, and the patient complained of a horrible taste in his mouth and fauces. After death, which occurred in thirteen hours, the membrane of the tongue was found destroyed (Flandin, op. cit. i, 495). Arsenic was detected in the stomach, the mucous coat of which was destroyed, or reduced to a gelatinous pulp; but it is not stated whether it was mixed with corrosive sublimate or any other poison. This action on the mouth is very similar to that produced by corrosive sublimate. According to the reporter of this case, arsenic in a large dose corrodes and destroys the tissues with which it comes in contact; in his opinion it acts like an acid or a caustic substance (i, 557).

Solubility of arsenic.—The *solubility* of this substance in liquids is a frequent question on trials. The action of water is materially influenced by circumstances. I have found by numerous experiments (Guy's Hospital Reports, iv, 81), that hot water, cooling from 212° on the poison in powder, dissolves about the 400th part of its weight. This is in the proportion of nearly one grain and a quarter of white arsenic to about one fluid-ounce of water. Water boiled for an hour on the poison and allowed to cool, holds dissolved the 40th part of its weight, or about eleven grains to one ounce. Cold water allowed to stand for many hours on the poison, does not dissolve more than from the 1000th to the 500th part of its weight; *i. e.* one-half grain to one grain of arsenic to nearly one fluid-ounce of water. The presence of organic matter in a liquid renders the poison much less soluble. Thus, hot tea with milk and sugar, and cold porter, did not take up more than about half a grain to the ounce; while hot coffee and cold brandy did not dissolve more than one grain to the fluid-ounce. (Guy's Hosp. Rep. iv, 103.) Arsenic is soluble in most organic liquids, as milk, coffee, tea, wine, brandy, whiskey, and even oil. Although it is less soluble in these liquids than in distilled water, it is, nevertheless, taken up in sufficient quantity to occasion serious accidents, and even to destroy life. In 1845, a man was found guilty, at Madrid, of putting arsenic into oil with the intent to destroy his parents. Arsenic was found in the oil, and a portion of it given to a dog caused its death. (Journal de Chimie, 1845, p. 651.) Any alkali or alkaline carbonate dissolved in the liquid, greatly increases its solubility. Liquids, which are at all viscid or mucilaginous, such as gruel, arrow-root, cocoa, or syrup, may mechanically suspend the poison in almost any quantity, but in these cases it cannot be said to be dissolved. The solubility of arsenic was an important part of the evidence in the case of the Queen v. Hunter, tried at the Liverpool Lent Assizes, 1843. (See the medico-legal reports of this case which have been published, the one by Mr. Holland, and the other by Mr. Dyson of Manchester.) A medical witness must always take care to draw a distinction between the actual solution and the mechanical suspension of the poison in a viscid liquid, especially when it is necessary to determine whether the quantity taken was sufficient to kill. The case of Madeline Smith (ante, p. 187) involved a point of this nature. A doubt was raised whether eighty-eight grains of arsenic (found in the stomach) could have been taken unknowingly; and it was considered difficult to suggest a vehicle in which so large a dose could have been secretly administered. There is no doubt that this, or even a still larger dose of powdered arsenic, might be secretly administered in such liquids as gruel or cocoa.

Symptoms—Acute poisoning.—These will vary according to the form and dose in which the poison has been administered. The *time* at which they come on is generally in from half an hour to an hour after the poison had been swallowed. This is the average period. I have known them to appear in a quarter of an hour. Dr. Christison mentions an instance in which the symptoms began in eight minutes; in the case of Lofthouse, tried at the York Lent Assizes, 1835, the symptoms were proved to have attacked the deceased while he was in the act of eating a cake in which the poison was administered. In the case of Sager, quoted by Dr. Beck (Med. Jur. vol. ii, p. 546), it appears that the woman to whom the poison was administered experienced extreme distress immediately after she had taken it. It had been mixed with wine in which an egg had been stirred. This early occurrence of symptoms is very unusual, except in cases in which the poison is dissolved in some liquid. On the other hand, in an instance communicated to me by Mr. Todd, where one drachm had been taken on an empty stomach, no symptoms appeared for two hours; in one reported by Orfila, the symptoms did not show themselves for five hours; and in another which occurred to Dr. Lachèse, where a large dose was taken, the symptoms did not appear for seven hours. (Ann. d'Hyg. 1837, i, 344.) Dr. Thompson, of Liverpool, states that he met with a case in which from thirty to forty grains of arsenious acid, and the same quantity of chrome yellow, were taken. Symptoms of poisoning did not appear until five or six hours afterwards. (Med. Chir. Review, 1854, p. 294.) There may be every variety between these extremes. A case will be presently mentioned, in which their appearance was protracted for *ten* hours—the maximum period yet known (p. 325, post). A remarkable instance occurred to M. Tonnelier, in which the poison was taken by a young female at eleven o'clock in the morning, and no well-marked symptoms occurred for *eight hours;* there was then violent vomiting. After death a cyst, formed of mucous membrane, and containing arsenic, was found in the stomach; the poison having thus become sheathed over! (Flandin, i, 585.) (See, on this question, the case of Reg. v. Foster, Bury Lent Assizes, 1847.)

In a case communicated by Mr. Clegg to the Medical Times (Oct. 21, 1848), symptoms of violent irritation did not show themselves until twenty-three hours after the poison had been taken, and within about half an hour of the death of the patient. The girl was once sick shortly after having taken the poison, but the first decided symptoms were those of narcotism. The girl was a confirmed opium-eater, and this habit may in some measure have influenced the operation of the poison. From a case communicated to the Medical Gazette by Dr. W. Burke Ryan (vol. xlvii, p. 722), it appears that the active symptoms of irritation which commonly attend arsenical poisoning, may not show themselves until after the lapse of *nine hours* from the time at which the poison has been swallowed. With the exception of a case elsewhere recorded (post, p. 325), in which the interval was *ten hours*, this is the longest period of protraction on record. In other instances, there have been long intermissions. In all cases in which arsenic enters the system from without, as by its application to the skin, and to ulcerated or diseased surfaces, the symptoms are rarely manifested until after the lapse of several hours.

Their nature.—In an *acute* case of poisoning by arsenic the person first experiences faintness, depression, nausea, and sickness, with an intense burning pain in the region of the stomach, increased by pressure. The pain in the abdomen becomes more and more severe; and there is violent vomiting of a brown turbid matter, mixed with mucus, and sometimes streaked with blood. These symptoms are followed by purging, which is more or less violent; and this is accompanied by severe cramps in the calves of the legs. The matters discharged from the stomach and bowels have had in some instances a yellowish color, as it was supposed, from a partial conversion of the poison to sulphuret; but more probably from an admixture of bile. The vomited matters are in some cases colored by blood, or a mixture of blood and bile; they then present various shades of brown, or olive-green. The indigo used in coloring arsenic may give to them a blue color, or if mixed with bile, a green tint. The sooty arsenic renders them black. In other cases, the vomited matters present a milky-white appearance, consisting of flakes of mucus mixed with portions of white arsenic. The nature of the arsenic taken may be inferred from the color of the matter vomited. In the case of L'Angelier (Reg. v. Smith, Edinburgh, 1857), a witness deposed that the matter vomited by deceased, in the first stage of his illness, was a greenish substance of about the thickness of gruel. (Irvine's Report, p. 30.) Such would be the appearance produced by a mixture of blue arsenic and bile. Blue arsenic was in this case traced to the possession of the prisoner; but from an altered state of the bile, there may be green vomiting even when white arsenic has been taken. The vomiting is in general violent and incessant, and excited by any substance taken into the stomach. It brings no relief. There is tenesmus (straining), and the discharges by the bowels are frequently tinged with blood. There is a sense of constriction, with a feeling of dryness or burning heat in the throat, commonly accompanied by intense thirst. The pulse is small, very frequent, and irregular; sometimes wholly imperceptible. The skin is cold and clammy in the stage of collapse; at other times it is very hot, or there are rapid alternations of heat and cold. There is great restlessness. The respiration is painful from the tender state of the abdomen. Before death, coma sometimes supervenes, with paralysis, tetanic convulsions, or spasms in the muscles of the extremities. In one instance, trismus (lock-jaw) appeared in three-quarters of an hour. (Orfila, i, 449.) Such is the ordinary character of the symptoms in an *acute* case of arsenical poisoning, *i.e.* where from half an ounce to an ounce of the poison has been taken. As a general rule, the symptoms in the acute form of poisoning which proves fatal are *continuous*. Sometimes, however, there are remissions and even intermissions, which may lead to a deceptive hope of recovery, or (by the recurrence of symptoms) to an erroneous supposition that a fresh quantity of poison has been administered. In the case of the Duke of Praslin, who died from the effects of a large dose of arsenic, the remissions in symptoms, during the week which he survived, were such as to deceive the skilful physicians who attended him. At one time the vomiting had ceased, at another time the pain; the most persistent effects were the smallness and irregularity of the pulse and coldness of the limbs. (Ann. d'Hyg. 1847, vol. ii, p. 391.) Dr. Maclagan met with two cases in which there were intermissions of a prominent symptom (vomiting) for one and three days respectively. The symptoms recurred without, so far as could be ascertained, any fresh dose of poison being given to these persons. (Ed. Monthly Jour. Jan. 1853.)

The whole of the symptoms here described may not be met with in every instance. Thus the *pain*, which is usually excruciating, like a fire burning within the body, is sometimes absent. In a well-marked case of poisoning, which occurred in October, 1839, a dose of from one ounce to two ounces of arsenic was taken; there was no pain except of the most trifling character, just before death. It has been supposed that this symptom was absent when the dose was large; but a case occurred in Guy's Hospital, in 1836, in which only forty grains had been taken, and the patient died without complaining of pain. (Guy's Hos. Rep. iv, 68.) There are many similar instances on record. The symptoms of irritation of the stomach and bowels are seldom wanting, or there is vomiting, if there should be no purging. In one case of criminal poisoning by arsenic, in which I was consulted by Mr. Veasy, which was tried at the Bedford Spring Assizes, in 1842, there was neither vomiting nor purging. The quantity of poison taken must have been small. In a case which occurred to Dr. Feital, although half an ounce of arsenious acid had been taken, there was no vomiting (Med. Times, Dec. 12, 1846, p. 202). Intense *thirst* is a common symptom, but even this is sometimes absent. With respect to the urinary secretion there is no certain rule; it is sometimes suppressed, as in several cases reported by M. Flandin; at other times it is natural, or only slightly diminished. (Des Poisons, i, 521.) It is necessary for a medical jurist to attend to these anomalies, as otherwise the symptoms of arsenical poisoning may be easily mistaken for those of disease.

The following case is quoted by Belloc. A young woman, with the design of committing suicide, procured a lump of arsenic. She began by biting it; but, as she could not procure sufficient in this way, she broke it up into coarse fragments, put them into a glass of water, and swallowed them. This was in the morning, and she went the whole of the day without suffering any marked uneasiness. At six o'clock in the evening she was seen by M. Laborde,—and there were then no febrile symptoms. At eight o'clock she suffered from pain in the abdomen. At eleven o'clock she appeared to be more calm than ever, and had a strong desire to sleep. At three in the morning she sat up in her bed, complained a little of her stomach, and then died, without the least appearance of suffering. She vomited some fragments of arsenic before death. On opening the stomach, the vessels were found gorged, and there were coagula of blood in the folds of the mucous membrane at the larger end. There were marks of excoriation about the lips, mouth, and gullet. (Cours de Méd. Lég. 122.) No one acquainted with the usual effects of arsenic could have suspected this to have been a case of arsenical poisoning. The symptoms were probably more protracted in their appearance than in any other instance yet recorded, not having manifested themselves, as we may infer, until *ten hours* after the taking of the poison. They appear to have been at no time severe—there is no account of purging having been among them—they underwent a complete remission before death, and the deceased expired as from the effects of a narcotic, about seventeen hours after taking the poison.

It might be supposed, *à priori*, that the symptoms of irritation occasioned by arsenic, would be protracted in their appearance or mitigated in their character when the poison was taken mixed with opium; but in one well-marked case, in which a large dose of arsenic was swallowed with upwards of an ounce of laudanum, there was severe pain, abundant vomiting for two hours, and death took place in six hours. (Med.-Chir. Rev. vii, 170; also, Ann. d'Hyg. 1847, ii, p. 199.)

From this account of the symptoms produced by arsenic it will be seen that there is great difficulty in classifying cases. On referring to many which I have been enabled to collect, the symptoms do not appear to bear any relation to the *quantity* of poison or to the *form* in which it is administered. Those indicative of irritation in the stomach and bowels, or of an affection of the brain, have equally occurred, whether the dose was small or large, and whether the poison was in the state of powder or solution. The same observation may be made of other cases in which symptoms of an affection of the brain have followed those of irritation. Hence, it appears to me that any useful generalization is impossible, for the exceptions are so numerous as to show that the arbitrary divisions which must necessarily be made, can be of no practical utility. In the following case, which occurred to Dr. May, there was an entire absence of the prominent symptoms of arsenical poisoning: A child, aged 20 months, ate some paste consisting of honey, flour, and arsenic, prepared for destroying mice. He was caught in the act of eating it, and a considerable portion was forced from his mouth. Some ipecacuanha wine was given to him, and he vomited freely,—the ejected matter consisting chiefly of mucus of a yellowish color, with some of the paste suspended in it. The vomiting was encouraged, and milk was freely given. Between the fits of vomiting he appeared lively. In about two hours he had two natural motions, and was sleeping calmly as usual; he had had no pain; respiration a little hurried. In about six or seven hours he became somewhat restless, but there was no expression of pain. Soon after this, he became worse, the surface was cold, the lips were livid, eyes sunk, the pupils fixed and rather dilated, pulse scarcely perceptible, and respiration feeble, accompanied with sighing. After lying for half an hour in this condition, he expired without a struggle, nearly eight hours after taking the poison. On inspection, the stomach and intestines exhibited but little deviation from the healthy state. The stomach contained mucus and a portion of the paste, but the villous surface presented no signs of inflammation. (Prov. Med. Journ. July 16th, 1845, 453.) Dr. May very properly observes, that had not the child been seen to eat the paste, there was not a symptom, nor after death any morbid appearance, to indicate the true cause of the fatal illness.

It has been supposed that stupor and other symptoms of cerebral disturbance were more likely to occur when the dose of arsenic was large: but a case was communicated to the London Medical Review (April, 1811, p. 188), by Mr. Soden, of Coventry, which shows that, with a large dose of arsenic and rapid death, there may be violent symptoms affecting the stomach and bowels, and few or none indicative of nervous disorder. A man, aged 22, purchased seven ounces of finely-powdered arsenic, and swallowed, between seven and eight in the morning, not less than four and probably six ounces of the poison. In about half an hour he was found vomiting: there was severe pain in the abdomen, with a rapid pulse and slight convulsions of the legs. In two hours purging supervened, and there was constant inclination to pass urine: the pain in the bowels became almost intolerable, the convulsive motions of the limbs more frequent, and the pulse more feeble, but still very quick. According to the antidotal doctrines at that time prevalent, sulphuret of potash (potassium) was largely exhibited to him. He died in less than *four hours*, after a dreadful fit of convulsive laughter, his limbs becoming suddenly rigid (tetanus). In this case, there was neither stupor nor faintness, but there was severe pain, with convulsions. On inspection, the stomach was found highly inflamed, "the mucous coat looked as though it had been beautifully injected," and two ounces of arsenic were found in the cavity of this organ.

The following case was referred to me for examination by Mr. Carter of Newbury, in July, 1845. A female, æt. 22, swallowed a large dose of arsenic. She was immediately afterwards seized with intense thirst, severe burning pain, violent vomiting and purging continuing incessantly until death, which took place in *seven* hours. There were no nervous symptoms. The stomach and the whole of the intestinal canal were greatly inflamed. Arsenic was detected in large quantity, mixed with flakes of mucus, in the stomach, throughout the small intestines, and in the cæcum. (G. H. R. vol. iv, Oct. 1846, 458.) On the other hand, if severe symptoms of irritation of the stomach and bowels thus follow large doses, those indicative of an affection of the nervous system may be produced by comparatively small doses of the poison. I am indebted to Mr. Todd, coroner for Hants, for the particulars of the following case: A man, æt. 24, swallowed, about six o'clock in the morning, on an empty stomach, a drachm of white arsenic mixed with half a teacupful of water. He complained of the mixture having a rough taste, but he went to his work, and it was only the suspicion that he had made a mistake and swallowed poison, that induced him to go to the house of a surgeon, about two hours afterwards, for an emetic. He then appeared to be very well, and made no complaint; the surgeon could not detect about him any symptom of arsenical poisoning, and was inclined to disbelieve the man's statement. Vomiting was produced by sulphate of zinc, and he threw up some yellow-colored matter. He saw the man again in five hours: he was then sitting in a drowsy state, with a countenance expressive of great anxiety, a blue tinge on the hands, pulse very feeble, and there was profuse purging. He complained of *no pain*, and said that he wanted to go to sleep. Vomiting had ceased. He died in this calm state in the evening, about fourteen hours after he had taken the poison.

The mixed nature of these cases will also be obvious from the following, reported by Dr. Letheby: A girl, aged 19, swallowed two grains and a half of arsenic dissolved. Restlessness during the night, and slight pain, were the only symptoms experienced. In the morning she was sick, complained of great thirst, and the pain was more intense. Vomiting increased, followed by purging, the countenance was pinched, and the extremities were cold. From this state she rallied, and slept comfortably the following night. The next morning she was cold and drowsy, the pulse was scarcely perceptible, and she was passing into a state of incipient coma. In this condition she died about thirty-six hours after taking the poison. The stomach was pale and nearly empty, and its mucous coat was raised by a number of vesicles containing air. (Med. Gaz. xxxix, 116.)

Chronic poisoning.—Should the person recover from the first effects, and the case be protracted, or should the dose have been small and frequently administered, there will be inflammation of the conjunctivæ, with suffusion of the eyes, and intolerance of light,—a condition which is, however, often present with the early symptoms above described. In a case reported by Mr. Jeffreys, an adult female died *in three hours* after taking arsenic in a pudding served at dinner. There was no vomiting or purging. In two hours she was in a state of complete collapse, and at this time it was noticed that the conjunctivæ (the membranes of the eyes) were red. (Med. Times, Aug. 30, 1851, p. 229.) There is also great sensibility or irritation of the skin, accompanied by a vesicular eruption, which has been called "eczema arsenicale." Sometimes this has assumed the form of nettle-rash, or of the eruption attending scarlet fever, for which disease arsenical poisoning has been mistaken. Local paralysis, preceded by numbness, or tingling in the fingers or toes, and other symptoms of nervous disorder, are also very common consequences. Paralysis from arsenic is sometimes general, and affects both the upper and lower limbs. It may supervene on the cessation of symptoms of gastric irritation in cases of acute poisoning. It may be complete, or amount only to great weakness (Annuaire de Thérapeutique, 1858, p. 229.) The patient becomes emaciated, and sinks exhausted. Exfoliation of the cuticle and skin of the tongue, with the falling off of the hair, has likewise been witnessed. (Cases of the Turners, 1815, Marshall, p. 44, 119.) Salivation has been observed to follow, especially when small doses of the poison have been given for a length of time. (Med. Gaz. xvi, 790.) A well-marked case of this kind occurred to Mr. Jones, in which the effects produced by small doses of arsenic might have been mistaken for those of mercury. There was fœtor of the breath, with superficial ulceration of the gums and throat. (Med. Gaz. May 8, 1840, vol. xxvi, p. 266.) Strangury has also been noticed among the secondary symptoms. (Marshall on Arsenic, p. 44, 314.) From a statement by this author (op. cit. p. 111), it appears that there was a yellow or jaundiced state of the countenance in one of the cases reported by him. A similar state of the countenance was noticed by Dr. Thompson in the case of L'Angelier—a fact which gave rise to some discussion at the trial of Madeline Smith. (Irvine's Report, p. 51.) Dr. Geoghegan has observed, that in several cases of poisoning by arsenite of copper, jaundice was among the symptoms.

A well-marked case of *slow poisoning* by arsenic is recorded by Flandin. It illustrates one form of secret murder, and is well calculated to inspire caution in trusting to symptoms as evidence of disease. A woman put daily into the soup of her fellow-servant, a very small quantity of white arsenic in powder. Shortly after dinner this person was seized with vomiting, which led to the rejection of the food and poison before the latter had caused any serious mischief. As this practice was continued for about six weeks, the stomach became exceedingly irritable, there was pain in the bowels, and the woman became much emaciated. There was also spitting of blood, with such a degree of nervous irritability, that a current of air caused an attack of spasms and convulsions. When the patient found that she could not bear anything on her stomach, she left the place, and passed two months in the country. Her health was gradually re-established there, and she returned to resume her usual occupations. The prisoner, however, renewed her attempts; and, to make sure of destroying life, gave her one morning in coffee, a strong dose of arsenic in powder: violent vomiting ensued, and the poison was expelled with the breakfast. Arsenic was detected in the vomited matter, and the explanation of the cause of the long previous illness then became clear. Under proper treatment the patient recovered. (Op. cit. i, 510.) I believe this mode of poisoning to be not unfrequent; and it behooves practitioners to be exceedingly guarded in their opinions,—for the usual characters of arsenical poisoning are completely masked. The symptoms might be easily referred, as a result of careless observation, to chronic inflammation or ulceration of the stomach leading to perforation.

In the case of Reg. v. Sagar (York Lent Assizes, 1858), the symptoms and appearances in the deceased, which were plainly those of arsenical poisoning, were referred by one of the medical witnesses to "spontaneous inflammation of the bowels." The stomach, duodenum, and rectum were much inflamed, and arsenic was found in the liver and kidneys, as well as in the stomach and bowels. To remove this chemical difficulty, it was suggested that as the deceased had had a sore leg some months previously (which had healed), the arsenic might have entered her body by absorption from some quack ointment which she had applied to the leg, and remained there! The jury acted upon these ingenious medical suggestions and acquitted the prisoner of the charge.

The case of Mrs. Wooler (Durham Winter Assizes, 1855) conveys an important lesson in this respect. The suspicions of the medical attendants were confirmed, but at too late a period to save the life of this lady. There are many anomalous cases on record in which the symptoms have diverged so much from the ordinary course as to embarrass medical practitioners. For some of these I must refer to a paper by Dr. Ogston, Med. Gaz. vol. xlvii, p. 181.

There is one form of chronic poisoning by arsenic on which it will be proper to make a few remarks, as the real cause may remain wholly unsuspected. Arsenic is largely employed in this country, under the form of emerald-green (aceto-arsenite of copper), and of orpiment, in the manufacture of decorative papers with which the walls of sitting and bedrooms are covered. Some persons have suffered from symptoms of chronic poisoning by arsenic, in which no other cause was apparent than the continued respiration of the air of their rooms, charged probably at times with a fine arsenical dust. On examining the papers, they will be found in some instances loaded with arsenic, laid on in a rough and coarse manner, so as to be easily removable by friction. Arsenic is thus used in imparting a green tint to some of the most costly as well as the cheapest decorative papers. It is a practice fraught with danger in more respects than one, and under a proper system of medical police, it would not be permitted. In the kingdom of Prussia, the use of these papers is strictly prohibited. If there has not hitherto been much complaint on the subject, it may be attributed to the fact that the cause has not been suspected. Many obscure cases of illness, referred at the time to constitutional and other causes, may probably have been due to the effects of arsenical dust thus inhaled day and night by those who inhabited the rooms. Dr. Hinds, who suffered from the effects himself, has described two cases in which the prominent symptoms were prostration of strength, headache, thirst, loss of appetite—an inflammatory state of the conjunctivæ, with heat and dryness of the fauces. (Med. Times and Gazette, May 23, 1857, p. 521.) A portion of the paper of the room in which these persons lived was sent to me, and on examination I found in the green pigment spread over it, a large quantity of arsenic. These facts should at least be borne in mind in cases in which it is suspected that poison is being secretly administered to another. (See post, ARSENITE OF COPPER.)

In a case, communicated by Mr. Jones to the Provincial Journal (November 18, 1843, p. 127), the action of small doses of arsenic, administered medicinally, produced the well-marked effects of *slow* poisoning. The liquor arsenicalis was prescribed for a lady affected with cutaneous disease, in doses of five drops (= 1-24th of a grain) of arsenious acid, and fifteen drops (=1-8th of a grain), three times a day. After continuing these doses more or less for a month, she was found laboring under the following symptoms: obstinate purging, frequent griping pains in the bowels, with almost constant desire to act; considerable tenderness over the whole abdomen, which was distended; constant pain and nausea after taking food, and frequent vomiting; skin cool and dry; intense thirst; tongue clean and red, resembling raw beef; pulse 100, small and feeble; sense of constriction in the throat, and copious flow of saliva; she had some gastric cough, with frequent raking of the throat; and expectoration of a muco-purulent secretion mixed with blood. There was much pain and tenderness down the spine, with occasional muscular tremors; a crampy feeling of the lower limbs, with partial loss of motion and sensation; they were swollen, of a livid color in places, and showed a tendency to slough. There was great emaciation; want of sleep, owing to the irritable state of the bowels; motions white, watery, and frothy; urine scanty, high-colored, and passed with an effort. From this account of the symptoms, it is obvious that a case of slow poisoning by arsenic might very easily be mistaken for gastro-enteritis, and treated accordingly. (See Ann. d'Hygiène, 1837, vol. i, p. 347.) A case of this masked form of arsenical poisoning occurred to Dr. Pfeufer. There was general and well-marked paralysis of the whole of the muscular system, and a complete loss of sensation in the fingers. It was only after a year that the patient began to recover the use of his limbs. It appeared that his wife had been in the habit of giving him small doses of arsenic in his food. None of the articles of food, or of the matters vomited, could be procured for analysis; nevertheless, the evidence of chronic poisoning from symptoms was considered to be sufficiently strong to justify a conviction. (Zeitschrift für Rationelle Medizin, B. vi, H. i, 102, Heidelberg, 1847.)

A case has been published by Dr. Hooper, which is of some interest in reference to this question (Med. Times, August 21, 1847): A gentleman, æt. 54, afflicted with palsy, was recommended by his physician to take arsenic medicinally. He took five minims of Fowler's solution three times a day, and continued this practice from October to the 24th of the following June, when he consulted Dr. Hooper respecting an attack of ophthalmia. He was at once ordered to discontinue the arsenic. Dr. Hooper calculated that, in eight months and a half, this gentleman had swallowed, in small doses, no less than *sixty-four grains* of arsenious acid! *i. e.* enough to kill thirty adult persons. The poison was probably eliminated so rapidly, that but a small quantity was retained at any one time within the system. It was quite sufficient, however, to produce that peculiar train of symptoms indicative of *chronic* poisoning by arsenic: *i. e.* headache, drowsiness, nausea; an inexpressible feeling of languor and prostration; pulse 90 to 100; gradually increasing paralysis; muscular tremors affecting the lower jaw, back, neck, and arms; inflammation of the eyes, accompanied by œdema of the conjunctivæ and erythema (inflammatory redness) of the face; want of sleep, with irritation of the throat, larynx, and trachea. Under these symptoms, the patient gradually sank and died on the 13th September following. (See, also, Prov. Med. Jour. Nov. 18, 1843.) We have here all the characters of slow poisoning by arsenic, indicated by wasting fever and general derangement of the bodily functions. The effects of the Aqua Toffana were very similar to these. Individuals, to whom this liquid was administered in small doses, died without the slightest suspicion of the cause of death being excited.

Arsenic not an accumulative poison.—It is well known that arsenic is carried into the circulation by absorption, and it is an important question regarding its medicinal use, whether its elimination in the living body takes place with the same rapidity as its absorption. I am not aware of any facts, which show that arsenic can be taken in non-fatal (medicinal) doses for a certain period, accumulate in the body, and then suddenly give rise, without increase of dose, to all the marked symptoms of acute poisoning. On the contrary, all experience is in favor of the rapid elimination of this poison; and, unless the doses are too frequently repeated, or too rapidly increased, no danger will accrue from the quantity administered.

Dr. Wilks informed me (May, 1858), that, in treating a case of eczema in a boy, he prescribed the twenty-fourth part of a grain of arsenic three times daily, making one-eighth of a grain per diem. This was continued for seventy days, so that in ten weeks the boy had taken nearly *nine grains* of arsenic, or a quantity sufficient to destroy four adults. I found by analysis, that arsenic was daily eliminated in the urine; and after the medicine was discontinued, it still continued to pass away by this channel for about ten days, when the boy left the hospital. Arsenic may remain in the body for a variable period after the administration of it has ceased; but there is no evidence that it so accumulates in the viscera as to be suddenly productive of dangerous symptoms; and experience shows that, if its use be discontinued, the viscera, after a few weeks, do not contain a trace of the poison. (See ante, p. 45.)

A medico-legal question in reference to this property arose in the case of Lacoste. From the presence of arsenic in the body, and from the symptoms under which deceased died, it was alleged that his death had been caused by poison. The quantity of arsenic found in the liver was about the 1-13th of a grain, and a still smaller proportion in the other organs. In defence, it was alleged that deceased was in the habit of taking Fowler's solution (of arsenic) daily as a remedy for skin-disease under which he was laboring. The Procureur-Général admitted this, but contended that, as the man was in the habit of taking arsenic medicinally, it was very improbable that he should have taken an overdose (a fatal dose); and it could not, therefore, be supposed that he had died through his own imprudence. The President inquired whether innocent doses of arsenic could possibly accumulate in the body and suddenly occasion violent (acute) symptoms and death. M. Devergie replied in the negative, M. Flandin (for the defence) in the affirmative—the latter resting his opinion not upon any fact observed in the human body, but upon the assumption that the elimination of this poison might not always go on, *pari passu*, with its absorption. (Flandin, Des Poisons, i, 484.) The opinion then given was based upon experiments on animals. M. Flandin states that he has confirmed it by observations on the human subject made since the trial. The facts, however, are not stated; but he quotes the opinion of an English writer that Fowler's solution should not be long continued; because arsenic, like lead, might accumulate in the system and ultimately produce paralysis. The woman Lacoste was acquitted of the murder; it was justly considered that the small quantity of arsenic found in the body might possibly have been derived from that which had been taken medicinally. (See ante, p. 50.) The case of slow poisoning related at p. 328, is decidedly adverse to the view that arsenic is an accumulative poison in the sense here implied. MM. Danger and Flandin gave to animals gradually increasing doses of arsenic in powder, mixed with their food. In beginning with small fractional parts of a grain, they found after an interval of nine months, that the animals might be made to take upwards of *fifteen grains* of arsenic in twenty-four hours without any influence on their appetite or health. During this time their urine was frequently analyzed at different intervals without any arsenic being detected. Three days after the last dose was given, the animals were killed, and their organs successively analyzed; but no arsenic was detected in the viscera, flesh, or bones. (Op. cit. i, 737.) M. Flandin thus appears to have conclusively proved by his experiments, contrary to his own opinion, that arsenic certainly does *not* accumulate within the body!

This inference may also be fairly drawn from the facts detailed in Dr. Hooper's case (ante, p. 330), and in numerous other instances in which arsenic has been employed for many weeks in medicinal doses. There is no evidence that the poison accumulates in the system; and the occurrence of violent symptoms of vomiting and purging would, in general, justify the inference that another and larger dose of poison had been taken shortly before. Mr. Hunt considers that arsenic used medicinally does accumulate in the body. (On the Skin, 1847, p. 11.) The facts which he adduces, however, in support of this view, admit of another explanation. The speedy elimination by the kidneys proves that the poison is thrown out of the system; and where any morbid changes affect these or other eliminating channels, symptoms of poisoning may of course show themselves.

Effects of external application.—Arsenic, it is well known, when applied to wounded or ulcerated surfaces, becomes absorbed, and produces the usual effects of poisoning. A case is reported in Rust's Magazine, in which a man covered his head with arsenic in powder to act as a depilatory. He was affected with the usual symptoms of arsenical poisoning, excepting purging, and he died on the *twentieth* day. The interior of the stomach, as well as the lower part of the gullet, was generally inflamed. The following case, communicated to me by Mr. Tubbs, proves that arsenic, when rubbed on the skin, has decidedly a local *irritant* action. A man, who was subject to piles, was in the habit of anointing himself with lard. By mistake, on one occasion, he used some white ointment containing arsenic. The next day he complained of an intolerable itching of the anus and scrotum; and, on examination, the parts were covered with pustules surrounded with an inflamed base. They resembled those which are produced by arsenic. On examining the matter from the pustules, it was found to contain arsenious acid. Frictions of lime-water and oil were used, and the patient soon recovered.

Instances of arsenic thus destroying life when applied externally, are by no means unfrequent. Two cases of its operating fatally, in children, when applied to the skin of the head for tinea capitis, will be found in the Annales d'Hygiène, 1830, ii, 437. In both, the mucous membrane of the stomach was found inflamed, and in one extensively. Dr. Stillé quotes the following case: A woman rubbed half an ounce of arsenic mixed with gin into the heads of her children, who were affected with porrigo (scalled head). This application was followed by redness and swelling of the face. One child, two years of age, died from the effects, having suffered from purging with paralysis of the lower limbs before death. There was no local inflammation produced. (Med. Jur. 1855, p. 420; Am. Jour. Med. Sci. July, 1851, p. 259.)

A trial took place (Reg. v. Port) at Chester Winter Assizes, 1844, in which a man, pretending to cure cancer, was charged with the death of a female, by the application of an arsenical plaster, as it was alleged, to the breast. The woman died in a fortnight. No satisfactory evidence was obtained of the symptoms during life, except that there had been vomiting; and the accused had taken care to remove the plaster so soon as serious symptoms began to appear,—hence, there was no direct chemical evidence of the nature of the substance actually employed. Dr. Brett, of Liverpool, detected absorbed arsenic in the substance of the stomach, liver, and spleen: the whole quantity detected was less than a *quarter of a grain!* The gullet, stomach, and intestines were found extensively inflamed. In January, 1845, a man in this city died apparently from the effects of arsenic absorbed through the skin of the arm. He was engaged in the manufacture of candles, to which arsenic was added in large proportion, and it was supposed that an abrasion of the skin had facilitated the absorption of the poison. The medical opinion given at the inquest, was decidedly that the deceased had died from the effects of arsenic thus introduced into the system. M. Flandin states, that on one occasion he examined the viscera of a woman who had been killed by the application of an arsenical powder for the cure of a scirrhous breast. The arsenic (absorbed) was discovered in various parts of the body, but especially in the liver, which contained as much as is usually found when the poison has been swallowed (Des Poisons, i, 230): the quantity was greater than that separated from all the other organs together. This case presents many points of interest. The poison did not begin to produce its well-marked effects until after the lapse of about *ten hours*. Death took place in about six days, and the urine was suppressed throughout. The mucous membrane of the stomach and intestines was in its natural state: in the duodenum it was slightly swollen or thickened. (Flandin i, 502.) The powder used by quacks as an application to scirrhous breasts is commonly a compound of arsenious acid, realgar (red arsenic), and oxide of iron. In this instance it was formed of 75 parts of the first-mentioned substance, and 25 parts of the two last. The quack stated that he did not apply more than four or five grains. MM. Chevallier and Bayard have given the details of two cases which proved fatal, owing to the application of arsenical compounds to the breasts of females. (Ann. d'Hyg. 1846, ii, p. 131.)

The symptoms are slower in appearing on these occasions than when the arsenic is swallowed, probably from the diseased surface being less absorbent than the mucous membrane of the stomach; but in an experiment performed by Mr. Swan, in which arsenious acid was introduced into a wound on the back of a dog, vomiting came on in two hours, and the animal died in six hours. The mucous membrane or inner coat of the stomach and intestines was found inflamed. (Action of Mercury, p. 33, 1847.) In a case somewhat similar to that above related, M. Flandin discovered arsenic in the breast to which the substance had been applied, but in no other part of the body. (Op. cit. i, 550.) Some facts regarding this form of poisoning will be found in the Ann. d'Hyg. 1846, ii, 131.

The destruction of life by the local application of arsenic to ulcerated surfaces, has never, so far as I am aware, been resorted to by criminals. It is obvious, that when death is not a consequence, serious injury to health may ensue, and life might be endangered. The external application of orpiment in the form of ointment has caused death. (See SULPHURETS OF ARSENIC, post.) Belloc states that he employed arsenical compounds externally to scirrhous tumors of the eye, cheek, and nose, without any serious consequences resulting. (Cours de Méd. Lég. 121.)

Several cases are reported in which arsenic has acted as a poison through the *unbroken skin*. Some of them are of old standing, and do not appear to have been very accurately observed. (Flandin, i, 542.) If the arsenic is in solution, it may become speedily absorbed: but when in powder, absorption would take place much more slowly. It is well known that comparatively insoluble substances may be introduced into the system by frictions through the skin (the endermic method), and arsenic does not appear to present any exception to this mode of operation. The thin skin of the human subject appears to absorb the poison more readily than the hard thick skin of animals; but M. Flandin found that dogs were speedily killed when arsenical ointments were rubbed upon the skin of the abdomen, or on the inside of the thighs. (Op. cit. i. 544.) All the symptoms of arsenical poisoning, although not appearing for two or three days, have been witnessed in the human subject in those cases in which powdered arsenic has been used as a depilatory. (See ante, p. 35.)

The effects of arsenic on the unbroken skin was a subject of inquiry at the trial of Miss Smith (Edinburgh, 1857). The reader will find some remarks in reference to this at p. 97, ante. (See also Ann. d'Hyg. 1846, ii, p. 157.) Accidents frequently occur from the use of arsenic by shepherds for the purpose of destroying the fly in sheep. Two shepherds were engaged in sheep-dipping for nine hours—the liquid used being a mixture of white arsenic in a solution of carbonate of potash. On the following day both men were attacked with similar symptoms. One of them when seen, on the fourth day from the dipping, had the skin of the scrotum covered with eczema rubrum, resembling the appearance after vesication and separation of the cuticle in patches. There had been also vesicles on the thighs. There was slight febrile disturbance, with intense thirst, which no drink could allay. In a few days the man recovered. (Lancet, Sept. 12, 1857, p. 282.) Dr. Watson, who relates this case, states that the man had previously had similar eruptions from the use of the dipping composition. Other shepherds, he ascertained, who had used arsenic, had also suffered from eruptions, principally on the hands, forearms, scrotum, and thighs; and this had happened when the arsenic was used alone. (See ARSENITE OF POTASH, post.)

It appears that in India, yellow arsenic is much employed in the manufacture of shell-lac, and it is not unusual for eruptions of an eczematous nature to appear on the skins of those who are engaged in the manufacture. (Dr. N. Chever's Med. Jur. for India, p. 584.)

APPEARANCES AFTER DEATH.—The striking changes produced by arsenic are generally confined to the stomach and intestines. They are commonly well marked in proportion to the largeness of the dose and the length of time which the individual has survived after taking the poison. Our attention must first be directed to the *stomach*. Arsenic seems to have a specific effect on this organ; for, however the poison may have entered into the system, whether through a wounded or ulcerated surface, or by the act of swallowing, the stomach has been found inflamed. Inflammation of this organ cannot, then, be always considered to depend on a local irritant action of the poison. The mucous membrane of the stomach, which is often covered with a thick layer of mucus, mixed with blood, and with scattered white pasty-looking patches of arsenious acid, is commonly found red and inflamed; the color, which is sometimes of a dull or brownish red, becomes brighter on exposure to the air, at other times it is of a deep crimson hue, interspersed with black-looking streaks or patches of altered blood. The redness is usually most strongly marked at the greater extremity; in one case it may be found spread over the whole mucous surface, giving to it the appearance of red velvet,—in another it will be chiefly seen on the prominences of the folds (rugæ). It frequently assumes a dotted or striated form, stretching in curved lines between the two openings of the stomach. Blood of a dark color is effused in various parts between the folds, or beneath the lining membrane, an appearance which has been mistaken for gangrene. (See ante, p. 210.) The stomach often contains a mucous liquid of a dark color tinged with blood. The coats are sometimes thickened in patches, being raised up into a sort of fungous-like tumor, with arsenic imbedded in them: at other times they have been found thinned; and in other cases of a gelatinous consistency and appearance. The mucous membrane is rarely ulcerated, and still more rarely gangrenous. Among numerous inspections, I have not seen one instance in which gangrene of the coats of the stomach had resulted from the action of arsenic. In general, death takes place before gangrene is set up. In the case of the Duke de Praslin, who died from the effects of arsenic on the seventh day, a gangrenous state of the coats of the stomach is said to have been observed. Between the two apertures of the stomach there were seven large eschars—they were black, with yellowish-white margins, and of a different consistency from the adjoining coats. They were in some parts horny. Around these gangrenous spots, which did not involve the whole substance of the coats, the mucous membrane was somewhat softened and of a deep red color, evidently due to inflammation. (Ann. d'Hyg. 1847, vol. ii, p. 396.)

Perforation of the stomach, as a result of the action of arsenic, is so unusual an appearance, that there are but three accurately reported instances on record. In a case examined by M. Chevallier, the stomach of a person who died from the effects of arsenic was found perforated at the larger end. The aperture is described to have been of the size of a franc-piece, round, soft, and somewhat thickened at its margin. There was no redness or sign of erosion about it, and there was no appearance of ulceration on other parts of the mucous coat. Externally the stomach was covered with false membranes, arising from inflammation of the peritoneum. (Ann. d'Hyg. 1852, vol. i, p. 448.) This case is so imperfectly reported that it is impossible to say whether the perforation was caused by arsenic, or whether it was the result of other morbid changes.

The mucous glands of the stomach have been found enlarged; but this is by no means an unusual morbid appearance from any cause of local irritation, without reference to poisoning. Various morbid appearances are said to have been met with in the lungs, heart, brain, and urinary organs; but they do not appear to be characteristic of arsenical poisoning. It is undoubtedly to the stomach and intestines that a medical jurist must look for the basis of medical evidence in regard to the appearances after death. In a case which I saw in June, 1857, in which a person died in twenty hours after he had taken two teaspoonfuls (300 grains) of arsenic, there was inflammation of the gullet as well as of the stomach, duodenum, and rectum. The mucous membrane had a reddish-brown color. At the pyloric end of the stomach, as well as in the duodenum, there were several black patches from effused and altered blood. There was congestion of the brain and its membranes, with enlargement of the liver; but these appearances were not referable to the action of arsenic. A small quantity of arsenic was found in the thick reddish fluid found in the stomach; but the greater part of the large dose taken had been thrown off by vomiting.

Period required for inflammation.—A witness is often asked in a court of law how long a time is required after the taking of the poison, for the production of these well-marked appearances in the stomach, more especially of *inflammation* of the mucous membrane. In reference to this question, we have the following facts. In a case which I was required to examine, a large dose of arsenic had been taken; the man, aged 21, died in *five hours*, and the stomach was found intensely inflamed, especially about the greater curvature. In a case that occurred to Mr. Thompson, of Nottingham, half an ounce of the poison was taken; the patient died in *six hours*, and the stomach was found uniformly red and inflamed. In another that occurred to Dr. Booth, of Birmingham, the same quantity of arsenic was taken; the patient died in *six hours and a half;* on inspection, the gullet was inflamed, the whole internal surface of the stomach was of an intense scarlet color, and there were redness and increased vascularity of the small intestines. In three cases of poisoning by arsenic which occurred to Mr. Foster, of Huntingdon, death took place in one, a child, at the end of *two hours;* in the second, an adult, at the end of *three hours and a half:* and in the third, after the lapse of about six hours. In each of these, the stomach was found highly inflamed, and in the one that proved fatal in two hours, the mucous membrane had a vermilion hue. This last I believe to be the shortest period at which inflammation of the stomach, from the effects of arsenic, has hitherto been seen in the human body.

Period required for ulceration.—Another question put to a witness may be this,—What period of time is required for *ulceration* of the mucous membrane to take place, as an effect of this poison? If arsenic has destroyed life with unusual rapidity, and the stomach is found ulcerated, an attempt may be made to refer this ulceration to some other cause. Such an attempt was made in the case of Rhymes, which was the subject of a criminal trial at Reading, in 1841. (Guy's Hospital Reports, Oct. 1841, p. 283.) I found ulceration of the mucous membrane, which had been completely removed in patches, although the deceased had survived the effects of the poison only *ten* hours. The deposition of the arsenic in and around the ulcers, as well as the appearance of recent inflammation about them, left no doubt that they had been produced by the poison, and were not owing to previous disease, as it was attempted to be urged in defence. When no arsenic is found in the stomach, a defence of this kind will carry with it considerable plausibility. In Waring's case a medical witness was questioned upon this point. The deceased was stated to have died from the effects of arsenic in *four hours;* the coats of the stomach were found ulcerated, but no poison could be detected in the organ. The witness admitted, on cross-examination, that it was contrary to experience that ulceration should be occasioned by an irritant poison in so short a time as four hours; but he nevertheless contended that this was the true cause. On such points, we can only be guided by observation; and one case of this kind is sufficient to place the possibility of ulceration being produced by arsenic, within a few hours, beyond all question. Dr. Christison mentions a case observed by Mr. Hewson, where many eroded spots existed on the stomach, although the person died from the effects of arsenic in *five* hours. (On Poisons, p. 340.)

Absence of inflammation.—But are the stomach and intestines always found inflamed in cases of poisoning by arsenic? The answer must be decidedly in the negative. Dr. May's case (ante, p. 326) furnishes a striking example of the occasional absence of inflammation. At the trial of McCracken, at the Derby Autumn Assizes, in 1832, for killing his wife with arsenic, the fact of poisoning was clearly established, and a large quantity of arsenic was found in the stomach of the deceased; but there was no appearance of inflammation, either in this organ or the intestines. The two following cases are recorded in Rust's Magazine. A servant-girl had some arsenic administered to her in chocolate. She was seized with nausea and violent pain in the stomach, and died the same evening. On inspection, there was no remarkable redness or inflammation of the stomach; but arsenic was found in the duodenum. A man was taken ill with vomiting and violent pain in the abdomen after partaking of some soup, and he died from symptoms of poisoning. On inspection, the mucous surface of the stomach presented no morbid change, with the exception of slight redness about the cardia. Arsenic was found in the contents of the intestines. In a case quoted by Flandin from Etmuller, a girl swallowed a large dose of arsenic, and died twelve hours afterwards, without having vomited, or manifested any symptoms. On inspection, arsenic was found in the stomach, but there was no material change in the organ. (Op. cit. i, 284.) In a case tried at the Hertford Lent Assizes, 1855 (Reg. v. Newton), the coats of the stomach were thickened and pulpy, but were entirely free from inflammation. Death was clearly caused by arsenic. In another case, the mucous membrane of the stomach was found so pale that, at first, death from poisoning by arsenic was not suspected. Even with symptoms of irritation of the stomach, well-marked appearances may be wanting. (See Dr. Letheby's case, ante, p. 327.) Occasionally the appearances are so slight, that were not the attention of the examiner specially directed to the fact of poisoning, they would be passed over. (See case by Dr. May, ante, p. 326.) These exceptional cases appear to show, that arsenic does not exert any local action of a chemical nature, like a corrosive, on the stomach; for the action of corrosives takes place on mere contact, without reference to the state of constitution, or the quantity of poison taken. Medical evidence of poisoning from appearances after death is in such cases entirely wanting; they are not very common, but still their occurrence proves, that unless great care be taken in forming an opinion, a case of arsenical poisoning may be easily overlooked. They teach this important fact in legal medicine, that the non-existence of striking changes in the alimentary canal after death, is no proof that the party has not died from the effects of arsenic. When the dose of arsenic is small, well-marked changes in the body are rarely met with.

In some instances, the mouth, pharynx and *gullet* have been found inflamed, but in general there are no appearances in this part of the alimentary canal to attract particular attention. The mucous membrane of the *small intestines* may be inflamed throughout, but commonly the inflammatory redness is confined to the *duodenum*, especially to that part which joins the pylorus. Of the large intestines, the *rectum* appears to be the most prone to inflammation. The liver, spleen, and kidneys present no appearances which can be considered characteristic of arsenical poisoning, although these, like the other soft organs of the body, are receptacles of the absorbed poison. It is worthy of remark, in relation to the known antiseptic properties of arsenic, that the parts specially affected by this poison (the stomach and intestines) occasionally present the well-marked characters of irritant poisoning for a long time after death. This was established in the case of the Queen v. Dazley, tried at the Bedford Summer Assizes, July, 1843. The prisoner was convicted of poisoning her husband with arsenic, upon evidence obtained by the exhumation and examination of the body six months after interment. The stomach and intestines were the only parts of the body undecomposed. The case presents many important subjects for reflection to the medical jurist; as, for example, the substitution of arsenic for medicine,—the length of time after death at which good evidence may be obtained from the dead body,—the fact of another person laboring under symptoms of poisoning by arsenic, who had accidentally partaken of the supposed medicine—and lastly, the evidence from the death of an animal which had swallowed some of the matter vomited by the deceased. In two cases (Reg. v. Chesham) referred to me by Mr. Lewis, coroner for Essex, a deep red inflammatory appearance of the mucous membrane, immediately below a layer of sulphuret of arsenic, was well-marked, although the bodies had been buried *nineteen* months. In a case which occurred in March, 1848, the stomach was also well preserved; and it retained an inflammatory redness after the lapse of *twelve* months. Absorbed arsenic does not appear, in all cases, to prevent the decomposition of the soft organs in which it is deposited. (See post.) For a summary of the appearances caused by arsenic, and its influence in modifying putrefactive changes, I must refer the reader to a paper by Dr. Geoghegan, in the Medical Gazette, vol. xlvi, pp. 171 and 218, and Observations on Arsenical Poisoning, Dublin Quarterly Journal, Feb. 1851.

FATAL DOSE.—According to Dr. Christison the smallest fatal dose of arsenic in powder was thirty grains. The man who took it died in six days. The smallest fatal dose in solution was four and a half grains, which killed a child of four years of age in six hours. (Op. cit. 295.) In a case that fell under my notice, I have reason to think that a young lady was killed by eating a portion of cake which could not have contained more than four grains of arsenic, and probably less than three grains. There is no doubt that very small doses of this poison are capable of producing serious effects; and that some constitutions may be more affected by such doses of arsenic than others. It is often safely given in medical doses of from 1-16th to 1-8th of a grain; but it is impossible to give half a grain without producing some of the symptoms of poisoning. Dr. Burne has reported the case of a young female, who took only the twentieth part of a grain daily for four days, making in the whole *one-fifth* of a grain of arsenic. Symptoms of inflammation of the stomach and alarming symptoms of a nervous character appeared, which endangered the patient's life, and rendered a discontinuance of the medicine absolutely necessary. She suffered from delirium, palsied shaking of the head, swimming of the eyes, and such a degree of debility and exhaustion as to place her in imminent danger. (Med. Gaz. xxv, 414.) Several facts of this kind are recorded by Dr. Lachése (Ann. d'Hyg. 1837, i, p. 349). Arsenic taken by a healthy adult in a dose of one-eighth of a grain may produce injurious effects: In a dose varying from a quarter to one-half of a grain the symptoms may assume the true character of poisoning. In November, 1857, I was consulted in reference to a case in which small doses of arsenic, given medicinally, produced sickness, irritation of the skin, and eczema over the whole body. The dose was here only 1-30th of a grain, once, or, in some cases, twice a day! The total quantity taken in seven days was only forty minims of Fowler's solution, or one-third of a grain of arsenic! The reader will find, under CHLORIDE OF ARSENIC (post), a case in which a healthy adult suffered from some severe symptoms of poisoning as a result of taking common medicinal doses. These facts show not only the highly poisonous nature of this mineral, but that in some persons there must be an idiosyncrasy or a special susceptibility to the poisonous action of arsenic. According to Dr. Elliotson this intolerance of arsenic manifested by the production of symptoms of poisoning from small medicinal doses is brought out by the previous use of tartarized antimony. (Med. Times and Gazette, May 22, 1858, p. 524.)

The following case occurred in London, in October, 1839. At a large dinner party, it was observed that three persons, who had partaken of the port wine on the table, were seized with symptoms of poisoning. The wine was suspected to contain poison, and it was sent to me for examination. It was clear, of the usual color, odor, and taste, and possessed all the characters of good wine; but there was a small quantity of a reddish-white sediment at the bottom of the bottle. From the account of the symptoms, the wine was suspected to contain arsenic: this was found to be the case, and the quantity of poison dissolved amounted to about 1·2 grain in each fluid-ounce. The following were the facts: A child, of about sixteen months, took a quantity of the wine, containing about *one-third of a grain* of arsenic. In twenty minutes this child became sick, vomited violently for three hours, and then recovered. A lady, aged 52, took a quantity of wine, containing rather less than *two grains* of arsenic. In about half an hour, she experienced faintness. Violent vomiting came on, and lasted four hours, but there was no pain. She then gradually recovered. A gentleman, aged 40, took a quantity of the wine, containing rather more than *two grains* of the poison. The symptoms in him were similar, but more severe; and had he taken another glass of the wine, it is probable that he would have been killed. It may be proper to observe, that, although this wine was perfectly saturated with arsenic, not the least taste was perceived by any of the parties.

This case shows that two grains of arsenic have been taken without causing death, but it is not thence to be inferred that two grains, or even less, may not suffice to destroy life. As Dr. Christison justly remarks, the two adults may have here owed their escape to the fact, that the poison was taken on a full stomach, and that there was violent vomiting; but this observation cannot apply to the child. From the symptoms produced, we shall certainly be warranted in asserting, that a dose of three grains is very likely to prove fatal to an adult. According to Dr. Lachèse, from one to two grains may act fatally in a few days. (Ann. d'Hyg. 1837, i, 334.) It is highly probable that this dose would prove fatal to a child, or to weak and debilitated persons. The smallest fatal dose hitherto recorded, was observed in a case communicated by Dr. Castle, of Leeds, to the Provincial Journal (June 28, 1848, p. 347). A woman took half an ounce of Fowler's solution (arsenite of potash), in unknown doses, during a period of five days. She then died; and, on examination, the stomach and intestines were found inflamed. Death took place by syncope (mortal fainting), and there was an absence of vomiting and purging. The quantity of arsenic, which here destroyed life, could not have been more than *two grains*. In a case, reported to the Pathological Society of London, by Dr. Letheby, two grains and a half of arsenic, contained in two ounces of Fly-water, killed a robust healthy girl, aged nineteen, in thirty-six hours. (Med. Gaz. xxxix, 116.) These facts will justify a medical witness in stating that, under circumstances favorable to its operation, the fatal dose of this poison is from *two to three grains*.

When the dose is below two grains, although the symptoms of poisoning may be violently developed, the person generally recovers. A remarkable illustration of this is furnished by a set of cases on which I was consulted in December, 1857. At a large Industrial school, near London, *three hundred and forty children* were suddenly seized with symptoms of poisoning by arsenic, soon after breakfast. They had been supplied with milk diluted with water from a boiler, into which a quantity of an alkaline solution of arsenic had been placed under the notion that the alkaline arsenite would effectually cleanse it of fur. Two gallons of this *cleansing* liquid, containing about *nine pounds of arsenic*, perfectly dissolved by the aid of a large quantity of soda, had been well mixed with water in the steam-boiler of the establishment, without any information being given of its dangerous properties! Fortunately only four gallons of the poisoned water were drawn out of the boiler. This quantity was mixed with thirty gallons of milk, and divided among the three hundred and forty children,—about a gallon of the mixture being assigned to ten children. Upon an average, each child took *a grain* of arsenic more or less. The nature of the poison was soon discovered, and proper remedies suggested and employed. It is remarkable that, in this wholesale poisoning, the symptoms varied but little among the children. There was shivering, with pain in the stomach and bowels, and, in most of the cases, vomiting of a clear, ropy, mucous fluid, of a green color (the cleansing liquid having this color). These symptoms were developed within one hour. In about three hours after the meal, pain in the forehead, more or less intense, was a prominent symptom, and there was a copious discharge of a watery mucous fluid from the nose (coryza). Seven had cough of a croupy character, three vomited blood, and one passed blood by the bowels. Some suffered from inflammation of the stomach: of these six only were under treatment at the end of the first week, and one did not recover until after the second week. The treatment consisted in giving gum-water with albumen, and in keeping up vomiting by emetics or warm greasy water for twelve hours: after this, castor-oil was administered. The whole of these children recovered, and thus this occurrence did not become a subject of public investigation. The persons, who used this dangerous article for cleansing boilers, were warned. These cases appear to show, that, except under peculiar circumstances, a dose of one grain of this poison is not likely to prove fatal. In the wholesale poisoning of the English, at Hong-Kong, by the introduction of arsenic into bread, there appears to have been an equally remarkable escape. Professor Rose, of Berlin, examined a portion of the poisoned bread, and found that it contained about two grains of arsenic to the ounce. The arsenic could not be mechanically separated from the bread, and there was no doubt that the poison had been mixed deliberately with the dough, as there was great uniformity in the mixture (Archiv der Gesundheitspflege, 1857, i, p. 8). The escape of those who partook of this bread, must have been due to early vomiting, as each ounce of bread would have contained a quantity of poison sufficient to destroy life.

Recovery from large doses.—Persons have recovered after having taken very large doses of this poison. M. Bertrand states, that he swallowed five grains of arsenious acid with impunity. (Christison, 862.) The poison was here mixed with a large quantity of charcoal. A case is reported, in which *sixty grains* were taken by a physician, who recovered without suffering very severely. (Med. Gaz. xi, 771.) In another instance, a person recovered after having taken half an ounce of arsenic. The stomach-pump was not used, and the arsenic appears to have been carried off by vomiting and purging. (Med. Gaz. xix, 288.) In a case, reported by Dr. Feital, it is said there was *no vomiting*, and yet the person recovered after having taken half an ounce! (Med. Times, Dec. 12, 1846, p. 202.) A case is quoted by Wibmer, from an American journal, in which a man is reported to have recovered, in three or four days, after having taken one ounce and a half of arsenic. There was violent vomiting. (Arzneimittel, i, 278.) Mr. Bryant has reported a case, in which a man recovered after having taken two tablespoonfuls, or upwards, of two ounces of arsenic. There was not much pain; the symptoms were rather those of a narcotic character. (Lancet, Oct. 2, 1852, p. 299; also, 1857, ii, p. 114.) Cases of recovery, when large doses have been taken, are not very common. They must be regarded as exceptions to the general rule. It would be in the highest degree improper to infer from them that a large dose of this poison may be taken with impunity. In these instances we commonly find, either that the arsenic has been taken on a full stomach, or, under appropriate treatment, that it has been speedily ejected by vomiting and purging.

PERIOD AT WHICH DEATH TAKES PLACE.—Full doses of arsenic commonly prove fatal in from eighteen hours to three days. Probably the average time at which death takes place is twenty-four hours. But the poison may destroy life within a much shorter period than this. There are now many authentic cases reported, in which death has taken place in from three to six hours. (Guy's Hospital Reports, Oct. 1850, p. 183.) I have met with a well-marked case of death from arsenic in five hours, and with another in four hours. (For a case fatal in five hours, see Ann.

d'Hyg. 1837, i, 339.) It is singular that a few years since observations were so limited, that it was thought to be impossible for arsenic to destroy life in a shorter period of time than seven hours! (See ante, p. 150, Russell's case.) This rapidity of death was actually considered as a medical fact, which, in some measure, tended to negative the allegation of death from arsenic! One of the most rapidly fatal cases on record, I believe, to be that which occurred to Mr. Foster (ante, 337). This gentleman satisfactorily ascertained that a child, under three years of age, died within *two hours* from the effects of arsenic. The quantity taken could not be determined; but the time at which death takes place, is by no means dependent on the quantity of poison taken. Dr. Borland, who formerly attended my lectures, communicated to me a case in which death probably occurred in less than two hours. A young married female, of a delicate nervous temperament, bought two ounces of arsenic. The whole, or the greater part of this, she swallowed dry, and washed it down with some milk procured on her way home. Dr. Borland inquired into the circumstances, and found that the period from the time of the deceased taking the poison until her death, must have been less than *two hours*. There was no vomiting, except of a little glairy matter, from artificial means employed, neither had the bowels been acted on from first to last, nor could it be ascertained that she had suffered much pain. Death took place quite suddenly. A case of poisoning by arsenic, but somewhat doubtful, is reported by Metzger to have proved fatal in *half an hour*. The patient died in convulsions. (System de Ger. Arzneim. 256.) In some of these instances of rapid death, the brain and nervous system have been observed to be affected, the patient suffering from narcotism and convulsions; but this by no means implies that symptoms of irritation are always absent. In Mr. Soden's case (p. 326), in which not less than four, and probably six ounces of the poison had been taken, the patient died in less than four hours, and two ounces of arsenic were found in the stomach. We have here an instance, which occurred in March, 1810, of arsenic destroying life and producing excessive inflammation in less than *four hours;* and yet at a criminal trial, sixteen years afterwards (Lewes Assizes, 1826), it was a debated question with some of the medical witnesses, whether it was possible for a person to die from the effects of arsenic in less than *seven hours*, and respectable medical authorities were actually quoted against this view! This case shows the extreme danger of a court of justice relying for medico-legal facts of this description, upon the personal experience of witnesses.

From a case published by Dr. Dymock, it appears that a girl, æt. 20, took two ounces of powdered arsenic, and died in less than two hours and a half afterwards. There were no comatose symptoms: the girl was sensible to the last, and she had vomited violently. The mucous membrane of the stomach was covered with bright patches of a scarlet color. (Ed. Med. and Surg. Journ. April, 1843.) In thirteen cases of poisoning by arsenic recorded by Dr. Beck, the smallest quantity taken was one drachm, and the largest two drachms. The shortest period for death was four hours, the longest two days. (Dubl. Med. Press, May, 1845.) In some of these rapid cases of death, especially in those of Mr. Foster and that of Dr. Borland, it is evident from the symptoms that the brain and heart had become affected within the very short period of two hours. It would be advisable that, in any cases of rapid death from arsenic, the soft organs and tissues should be hereafter examined, in order to determine the earliest period after the administration at which they become penetrated with the poison. This investigation has been hitherto omitted, and the omission unfortunately leaves a wide range for speculative medical testimony.

With respect to the effect of *quantity*, I have known one case prove fatal in fifteen hours where forty grains had been taken; and in another, where an ounce (twelve times the above quantity) had been swallowed, the patient did not die for seventeen hours. Both patients were females of about the same age. It is a common opinion that large doses only kill with great rapidity; but this is not uniformly observed. In one instance, two ounces of the poison destroyed life in three hours and a half; but in another case (Waring) a dose of four or five grains killed a person in four hours. In some instances, death does not occur until long after the average period. In one case in which an adult swallowed about half an ounce, death did not take place for *fifty hours*, and it is remarkable that there was an entire absence of pain. (Med. Gaz. vol. xlviii, p. 446.) In the case of the Duke de Praslin, one large dose was taken, but death did not occur until the *sixth* day. (Ann. d'Hyg. 1847, ii, 367.) In October, 1847, a man who had swallowed 220 grains of arsenic was admitted into Guy's Hospital. He died on the *seventh* day. It is obvious that a patient who recovers from the first effects of this poison may still die from exhaustion or other secondary causes many days or weeks after having taken it. In the case of Reg. v. McCormick, Liverpool Winter Assizes, the child died, as it appeared, from one dose of arsenic, after the lapse of twelve days. (Med. Gaz. xxxiii, 484.) It had partially recovered from the first effects. In the case of Dr. Alexander death took place on the *sixteenth* day. (Med. Times and Gazette, April 18, 1857, p. 389.) In one instance, already mentioned, arsenic was applied externally to the head, and the person did not die until the *twentieth* day (p. 333). The longest duration of a case of poisoning by arsenic which I have met with, is reported by Belloc. A woman, æt. 56, employed a solution of arsenic in water to cure the itch, which had resisted the usual remedies. The skin became covered with an erysipelatous eruption, and the itch was cured, but she experienced severe suffering. Her health gradually failed, and she died after the lapse of *two years*, having suffered during the whole of this period from a general tremor of the limbs. (Cours de Méd. Lég. 121.)

TREATMENT.—If vomiting does not already exist as a direct effect of the poison, sulphate of zinc should be exhibited, and its emetic effects promoted by mucilaginous drinks, such as linseed-tea, milk, or albuminous liquids. A dose of tartar emetic, or of sulphate of copper, may be given in place of sulphate of zinc. When none of these can be procured, powdered mustard, in a proportion of from one to two teaspoonfuls in a glass of warm water, or, failing this, a glass of hot greasy water, which may be procured in every household, should be administered at intervals. A saponaceous liquid, made of equal parts of oil and lime-water, has been given with benefit. While the oil invests the poison, the lime tends to render less soluble that portion of arsenic which is dissolved. The stomach-pump may be usefully employed; but unless the patient is seen early, remedial means are seldom attended with success. It is proper to examine occasionally by Reinsch's process (see post) the liquid vomited, or withdrawn by the stomach-pump. We may thus ascertain when the poison is entirely removed from the stomach. In the event of the arsenic disappearing from the stomach and the person recovering from the first effects, it will be necessary to examine the urine which is passed daily. Arsenic may be found in this secretion for two or three weeks, or longer. Four ounces of urine are commonly sufficient for this observation. It should be concentrated by evaporation, and examined by Reinsch's process.

I have known death to occur in a case in which it was found, on subsequent examination, that every particle of poison had been removed from the stomach; and there are many instances of recovery on record, in which the arsenic appears to have been early ejected by constant vomiting and purging. The recovery has, however, been commonly attributed to some supposed antidote. Mr. Tubbs has found that, conjoined with the use of the stomach-pump and emetics of sulphate of zinc, a mixture of milk, lime-water, and albumen is useful. Such a mixture is undoubtedly well-fitted to envelope the particles of arsenic, and sheathe the coats of the stomach from the irritant action of the poison. In the ordinary cases of arsenical poisoning, *i. e.* when arsenic is taken in the form of powder, no confidence can be placed in the use of the so-called chemical antidotes,—*hydrated sesquioxide of iron* (prepared in precipitating persulphate of iron by ammonia, and leaving the alkali a little in excess) or the *hydrate of magnesia.* They may serve mechanically to suspend the poison, and thus facilitate its ejection from the stomach; but in this respect they possess no advantages over albumen or other viscid liquids. The *hydrate of magnesia*, as recommended by Bussy (Annuaire de Thérapeutique, 1847, p. 291), may be prepared by heating carbonate of magnesia to a low red heat in a crucible. One part will then form a gelatinous hydrate with fifty parts of water. It may be given in milk or other demulcent liquids. It forms a comparatively insoluble arsenite of magnesia; but the arsenites which are insoluble in water are soluble in the acid mucous secretions of the stomach, and act as powerful poisons. (See post, ARSENITE OF COPPER.)

When arsenic has been taken in solution, the hydrated oxide of iron, if given in large quantity, will precipitate the poison in an insoluble form, and it may then become beneficial. A mixture of hydrate of magnesia and persulphate of iron may be used. The two chemical antidotes as they are called, are present in this mixture, and the sulphate of magnesia produced by decomposition tends to act on the bowels and expel the poison.

The oxide of iron appears to have no more effect on *solid* arsenic than so much powdered brick-dust, and to rest upon this as a neutralizer of the poisonous action of solid arsenic would be a delusion. Dr. Geoghegan noticed, in examining the stomach of a man who had died from arsenic, and to whom this substance had been freely given, that the white particles of poison were lying in the midst of the oxide. Casper made the same observation in the case of a youth who died from a dose of arsenic in twenty-four hours. About a pound of the oxide was found in the stomach after death, and beneath this, the white particles of arsenic could be seen by a lens lying upon the surface of the mucous membrane. (Handbuch der Ger. Medicin, 1857, i, p. 419.) It has been suggested that if a large quantity of oxide of iron has been used in the treatment, and arsenic is found in the stomach only in traces,—the poison may really have been an impurity in the oxide. (See ante, p. 194.) An objection of this kind may be met by the discovery of arsenic in the liver or other organs, and by the evidence derivable from symptoms and appearances after death.

Our treatment must be chiefly directed to the early and entire removal of the poison from the stomach and bowels by emetics and purgatives. Orfila has recommended that diuretics should be employed, in order to promote the secretion of urine, and thus favor the more speedy elimination of the poison from the system. It appears to me that there are some objections to this mode of treatment: it involves the necessity that the whole of the poison should pass into the blood for the purpose of elimination; and further, that it should not be carried off gradually, as would be the case if diuretics were not administered, but that the process of absorption should be expedited by creating a drain upon the kidneys. The danger, however, in the absorption of poisons, appears to arise less from the absolute quantity taken up by the blood, than the quantity admitted into the circulation at any one time. Further, poisons are not always or necessarily eliminated by one secretion. Sometimes they escape more readily by the skin, at others by the saliva. It does not therefore follow that the promotion of the urinary secretion would always, *pro tanto*, promote the expulsion of the poison. The researches of M. Flandin show that the quantity of arsenic which escapes by the kidneys is, in some cases, small. These appear to me to be serious objections to a plan of treatment which, to be efficient, implies the saturation of the whole of the blood with poison in as short a time as possible. When arsenic is entirely expelled from the alimentary canal, there can be no injury in the employment of diuretics; but the question would then be, whether the patient would not recover just as readily without them. So long as any poison remains in the stomach or viscera, diuretics are likely to do more harm than good; and when all the poison is expelled, they appear to be useless. The diuretic plan entirely failed in the experiments performed on animals by M. Flandin (i, 583; also p. 47, ante). The free use of emetics and quickly acting purgatives, *e. g.* castor oil, will remove more of the poison in a few minutes or hours, than would be eliminated by diuretics in as many days.

CHAPTER XXIII.

ARSENIC—CHEMICAL ANALYSIS—TESTS FOR ARSENIOUS ACID—REDUCTION-PROCESS—THE LIQUID TESTS—OBJECTIONS—SUMMARY OF THEIR VALUE—MARSH'S PROCESS—REINSCH'S PROCESS—ARSENIC IN LIQUIDS CONTAINING ORGANIC MATTER—DETECTION IN THE CONTENTS OF THE STOMACH—IN THE TISSUES—RELATIVE DELICACY OF THE PROCESSES—METHODS OF DETERMINING THE QUANTITY OF ARSENIC IN THE FREE AND ABSORBED STATE.

CHEMICAL ANALYSIS.

Arsenic as a solid.—In the *simple state*, white arsenic may be identified by the following properties: 1. A small quantity of the powder, placed on platina-foil, is entirely volatilized at a gentle heat (380°) in a white vapor. Should there be any residue it is impurity. If a small portion of the white powder be gently heated in a glass tube of narrow bore, it will be sublimed, and form a ring of minute octahedral crystals, remarkable for their lustre and brilliancy. Under a microscope of high magnifying power (250 diameters) the appearance of these crystals is remarkably beautiful and characteristic; one not exceeding the 4000th of an inch in size may be easily recognized by the aid of this instrument. The form is that of the regular octahedron, of which the sides are equal. The crystals are frequently grouped, or nucleated; the solid angles are sometimes cut off, and occasionally equilateral triangular plates are seen. The forms are various; but all are traceable to the octahedron. Crystals which do not exceed the 10,000th, or even the 16,000th of an inch in diameter, present these microscopical characters distinctly; and the 1000th part of a grain of white arsenic will furnish many hundreds of crystals visible under the microscope. Generally speaking, the smallest crystals are those in which the octahedral form is the best defined. 2. If a portion of the powder be introduced on a fine platina wire into the edge of the flame of a spirit-lamp, it will impart a steel-blue color, and evolve a white vapor. It will be observed, in these experiments, that white arsenic in vapor possesses no odor. 3. On boiling a small quantity of the powder is distilled water, it is not dissolved; but it partly floats in a sort of white film, and is partly aggregated in small white masses, at the bottom of the vessel. It requires long boiling, in order that it should become dissolved and equally diffused through water (p. 322, ante). This was a point of some importance in the case of Reg. v. Lever (Central Criminal Court, June, 1844). A question here arose, whether arsenic would float on tea. I have observed that the film formed on putting powdered arsenic into a vessel of cold water, remained for five weeks on the surface, notwithstanding the occasional agitation of the vessel. 4. On adding a few drops of a solution of potash to the mixture of arsenic and water, and applying heat, the poison is entirely dissolved, forming a clear solution of arsenite of potash. 5. The powder is soluble by heat in hydrochloric acid, and when a piece of bright copper is immersed in the solution, it acquires a dark iron-gray stain from the deposit of metallic arsenic. 6. When the powder is treated with a solution of hydrosulphuret of ammonia in a watch-glass, there is no immediate change of color as there is with most metallic poisons. On heating the mixture, the white powder is dissolved; and on continuing the heat until the ammonia is expelled, a rich yellow or orange-red film is left (sulphuret of arsenic), which is soluble in all alkalies, and insoluble in hydrochloric acid. This yellow compound is produced from the mixture by spontaneous evaporation. A solution of sulphuretted hydrogen colors it slowly, and leaves by evaporation the same yellow compound. 7. It is oxidized and dissolved when heated in strong nitric acid; and on evaporation to dryness on a sand-bath, it leaves a white deliquescent residue (arsenic acid), which, when dissolved in a few drops of water, produces a brick-red colored precipitate with a solution of nitrate of silver. 8. When the powder is heated in a tube with two or three parts of charcoal, or of a carbonaceous flux, it yields an iron-gray sublimate of metallic arsenic, which has an odor of garlic as it is evolved in vapor. This is called the "reduction test or process."

Reduction process.—The best reducing agent is soda-flux, obtained by incinerating neutral acetate or tartrate of soda, in a covered platina crucible. When the quantity of arsenic is from one-fourth to one-twentieth part of a grain, the tube employed for this experiment may be three inches long, and from one-eighth to one-sixth of an inch in diameter. When the arsenic is in still smaller proportion, a tube of smaller diameter should be used; or, as recommended by Berzelius, the closed end of the tube may be drawn out into a bulb with a narrow neck. The sublimate of metallic arsenic is then concentrated in the slender neck, instead of being diffused over a large surface of glass. Heat should be gradually applied, first to the glass above the mixture, and then to the mixture itself. As the alkali in the flux retains some arsenic, it has been suggested that a slip of charcoal should be employed in place of soda-flux when the quantity of arsenic is very small. I have, however, never found it necessary to resort to the use of charcoal. The reduction process with the soda-flux will be found sufficient for any quantity of arsenic that is visible to the eye.

During the application of heat to the mixture in the tube, there is a perceptible odor resembling that of garlic, which is possessed by metallic arsenic only while passing from the state of vapor to arsenious acid. This *odor* was at one time looked upon as peculiar to arsenic, but no reliance is now placed on it as a matter of medical evidence,—it is a mere accessory result. Many mistakes were formerly made respecting it. Thus, we find it stated to have been perceived under circumstances in which it could not have been produced! (Marshall on Arsenic, p. 90, ed. 1817.) It was not then known that white arsenic (arsenious acid) possessed no odor in the state of vapor.

In this experiment of reduction, there are commonly two sublimates or rings deposited in the tube; the upper ring has a brown color, and appears to be a mixture of finely divided metallic arsenic and arsenious acid, or, according to some chemists, a sub-oxide, more volatile than the metal. The lower ring is smaller, well defined, and of an iron or steel-gray lustre. This is pure metallic arsenic. In order to determine the *weight of the sublimate*, the glass tube should be filed off closely on each side of the metallic rings, and weighed; the sublimate may then be driven off by heat, and the piece of glass again weighed; the difference or loss represents the weight of the sublimate. These sublimates are remarkably light, and require to be weighed in a delicate balance. I found, in one experiment, a large sublimate to weigh no more than ·08 grains. By heating gently the piece of tube, reduced to powder in an agate-mortar, in another tube of larger diameter, the metallic arsenic, during volatilization, forms octahedral crystals of arsenious acid, which, after examination by the microscope, may be either dissolved in a few drops of water, and tested by the liquid tests (see post, p. 350), or submitted to the process mentioned under 6, supra. This process is as satisfactory as the application of the liquid tests, and it has the advantage of not leading to the subdivision and dilution of a small quantity of arsenic. One of the *metallic* rings should be also submitted to the action of nitric acid,—converted to arsenic acid, and tested by nitrate of silver, as under Exp. 6. Dr. Christison states, that by the reduction process a distinct metallic sublimate may be obtained from the 300th part of a grain of arsenic (op. cit. 260). These sublimates may be preserved unchanged for years by filing off the ends of the tube, and then hermetically sealing them in the flame of a spirit-lamp.

Objections to the reduction process.—The demonstration of the presence of arsenic is complete when all the results described under 8 are obtained. The other tests are useful, as indicating the properties of arsenic, but they are not necessary when metallic sublimates, convertible to octahedral crystals by heat, and to arsenic acid by the action of nitric acid, have been procured. With such evidence of the chemical nature of the sublimates as that above described, there are no reasonable objections to the reduction process. Cadmium, selenium, and mercury produce sublimates, but these do not possess the appearance or properties of the arsenical sublimate. Fixed stains in the glass tube from the presence of lead, or from adhering charcoal, cannot be mistaken for a volatile metallic deposit. Arsenic is sometimes used in the manufacture of glass, but the whole is volatilized during the process. (See Ann. d'Hyg. 1834, vol. i, p. 224.) It is contained as an ingredient in some kinds of opal glass; but this is not used for chemical purposes. The process of reduction, therefore, with the simple precautions above mentioned respecting the properties of the sublimate, is conclusive of the nature of the substance under examination.

Arsenic in solution in water: Liquid tests.—The aqueous solution of arsenic is clear, colorless, possesses scarcely any perceptible taste, and has a very faint acid reaction. In this state, we should first evaporate slowly a few drops on a glass plate, when a crystalline crust will be obtained. On examining this crust by a microscope, it will be found to consist of numerous minute octahedral crystals, presenting equilateral triangular surfaces by reflected light. By this simple experiment, arsenic is distinguished from every other metallic poison. 1. On adding to the solution, *Ammonio-nitrate of silver*, a rich yellow precipitate of arsenite of silver falls down: rapidly changing in color to an olive-brown.

This test is made by adding to a very strong solution of nitrate of silver, a weak solution of ammonia, and continuing to add the latter, until the brown oxide of silver, at first thrown down, is almost redissolved. The yellow precipitate is soluble in nitric, tartaric, citric, and acetic acids, as well as in a solution of ammonia. It is not dissolved by potash or soda. 2. On adding to the solution of arsenic, *Ammonio-sulphate of copper*, a rich green precipitate is formed (Scheele's green), the tint of which varies, according to the proportion of arsenic present and the quantity of the test added; hence, if the quantity of arsenic be small, no green precipitate at first appears: the liquid simply acquires a blue color from the test. In less than an hour, if arsenic be present, a bright green deposit is formed, which may be easily separated from the blue liquid by filtration, or decantation. This test is made by adding ammonia to a weak solution of sulphate of copper, until the bluish-white precipitate, at first produced, is nearly redissolved: it should not be used in large quantity if concentrated, as it possesses a deep violet-blue color, which obscures the green color of the precipitate formed. The precipitated arsenite of copper is soluble in all acids, mineral and vegetable, and in ammonia, but not in potash or soda. When collected and dried, it possesses this characteristic property: by very slowly heating a few grains in a tube of small bore, arsenious acid is sublimed in a ring of minute resplendent octahedral crystals, visible to the eye, or by the aid of a lens or microscope. *Objections.*—The tests above described are called the liquid tests for arsenic. The *Silver* test, first discovered by Mr. Hume, in 1789 (Marshall on Arsenic, 87), acts with remarkable delicacy, and is of use as a corroborative test in the various processes for determining the presence of this poison in the body. A solution of an *alkaline phosphate*, which yields a yellow precipitate with nitrate of silver, is not affected by the ammonio-nitrate when properly made; and conversely, a solution of arsenious acid gives only a faint turbidness with nitrate of silver, while it is copiously precipitated of a yellow color by the ammonio-nitrate. A diluted solution of *phosphoric acid* may be, in some cases, precipitated by this test, exactly like a solution of arsenic; but there is one general answer to these objections. We do not rely upon the application of *one* test, but of several; and the fallacies attending one are removed by the employment of others. With respect to the delicate reaction of the silver-test, Mr. Marshall states (On Arsenic, p. 94, ed. 1817), that it is fully capable of detecting the 1000th part of a grain in solution,—a proof that its application was well understood more than a quarter of a century ago. Dr. Traill asserts that the 16,000th part of a grain of arsenic in solution is precipitated by the silver-test, and that with the 10,000th part of a grain a precipitate is visible to the eye. I have found that the 8000th part of a grain dissolved in one drop of water, gave a pale-yellow film; but the result materially depended on the quantity of water present. Thus the 4000th part of a grain of arsenic, in ten drops of water, was not perceptibly affected by the test; but the 2000th of a grain, dissolved in four drops of water, gave a decidedly yellow precipitate. The evidence derivable from these minute reactions would not be of much value, except that the test is used to corroborate inferences from the results of other experiments.

The *Copper* test is far less delicate in its reaction, and having an intensely blue color, it entirely conceals the green tint which may be given by a small quantity of precipitated arsenite of copper; but, in spite of this, if arsenic is present, the green precipitate is, after some hours, deposited at the bottom of the vessel. In cautiously adding a solution of arsenic of known strength to a few drops of this test, no green tint was imparted, until the quantity of arsenic amounted to the 173d part of a grain in less than one fluid-drachm of water—the degree of dilution being about 8640 times. Whenever the arsenic is in small quantity in its aqueous solution, this should be concentratd to the smallest possible bulk, and not more than one or two drops of the ammonio-sulphate should be added by means of a glass rod.

No one, in the present day, would think of employing these liquid tests in solutions, in which arsenic was mixed with *organic matter*.[1] Almost all liquids used as articles of food, are precipitated or colored by one or both of them, somewhat like a solution of arsenic, although none of this poison is present. Thus, then, any evidence founded on the production of *color*, unless the arsenic is dissolved in pure water, or unless the precipitates be proved to contain arsenic, should be rejected. These liquid tests are now employed rather as adjuncts to other processes, than as a direct means of detecting the poison. An exclusive reliance upon them, as *color tests*, has led to the rejection of chemical evidence on several trials, where they had been most improperly employed in the analysis of suspected liquids containing organic matter. The trial of Donnall at Launceston, in 1817, affords a memorable lesson to the medical jurist on this subject. (Smith's Anal. of Med. Ev. p. 212.)

3. *Sulphuretted hydrogen gas. Sulphur* or *Gaseous test.*—The hydrosulphuret of ammonia gives no precipitate in a solution of arsenic until an acid has been added, whereby arsenic is known from most metallic poisons. On adding an acid (acetic or pure diluted hydrochloric) a rich golden yellow-colored precipitate is thrown down (orpiment or sesquisulphuret of arsenic). It is better, however, to employ in medico-legal analysis, a current of sulphuretted hydrogen gas, which is easily procured by adding sulphuret of iron to one part of strong sulphuric acid and three parts of water in a proper apparatus. The arsenical liquid should be slightly acidulated with acetic or very diluted muriatic acid, before the gas is passed into it: at least care should be taken that it is not alkaline. The yellow compound is immediately produced and dissolved if arsenic is present in small quantity: but it may be collected after boiling the liquid so as to drive off the surplus gas. This yellow precipitate is known to be sulphuret of arsenic by the following properties: 1. It is insoluble in water, alcohol, and ether, as well as in all acids, mineral (muriatic) and vegetable; but it is decomposed by strong nitric and nitro-muriatic acids, leaving on evaporation at a low temperature arsenic acid, which may be neutralized by ammonia and tested by nitrate of silver. A brick-red precipitate (Exp. 7, ante, p. 348), will indicate that the yellow compound was sulphuret of arsenic, or orpiment. If strong nitric acid alone be used there will be no risk of losing any portion of the arsenic. 2. It is immediately dissolved by caustic potash, soda, or ammonia, forming, if no organic matter be present, a colorless

solution. 3. When dried and strongly heated, with a mixture of three parts of well-dried carbonate of soda and one part of cyanide of potassium, it furnishes a sublimate of metallic arsenic. This sublimate may then be tested by the processes already described (ante, p. 349). 4. The precipitated sulphuret may be deflagrated in a porcelain capsule with a mixture of pure nitrate and bicarbonate of potash. The saline residue, acidulated with nitric acid, should be evaporated, and the arseniate of potash dissolved out by a small quantity of water. The nitrate of silver may then be added to this solution, when the brick-red arseniate of silver will be produced. Unless these properties are proved to exist in the yellow precipitate formed by sulphuretted hydrogen in an unknown liquid, it cannot be safely regarded as a compound of arsenic. On the other hand, when they are possessed by the precipitate, it must be arsenic, and can be no other substance. The sulphur test is extremely delicate in its reaction. It begins to give a yellow tinge when the liquid contains only the 4000th part of a grain of arsenious acid in ten drops of water; the arsenic therefore forming about the 40,000th part of the solution. This becomes more decided with the 2000th part of a grain, and still more with the 250th part of a grain: the sulphuret is not, however, actually precipitated until diluted hydrochloric acid, in which it is insoluble, has been added to the liquid. It is important to observe that the effect produced by this test will materially depend on the quantity of water in which the given weight of arsenic happens to be diffused. In one experiment the gas was passed into a solution containing the 400th part of a grain in twenty drops of water: the results were clear and decided; the liquid acquired a rich golden-yellow color, but when passed into a solution containing the same weight of arsenic in half an ounce of water, a yellow tint was scarcely perceptible. The arsenic in the first case was in the proportion of the 8000th, and in the second of only the 1,000,000th part of the solution. Dilution therefore seriously affects the chemical results. *Objections.*—Many objections have been taken on criminal trials to the medical evidence, founded on the application of this test; but it may be at once stated that there is no objection to the inference derivable from the sulphur test, provided the properties of the precipitate, under 1, 2, 3, or 4, have been determined. The objections apply only to those cases in which arsenic is said to be present, when a yellow precipitate is produced by sulphuretted hydrogen. 1. *Cadmium.* It is remarkable that this metal should furnish, at the same time, a plausible ground of objection, both to the process by reduction from the solid state, and to the gaseous test applied to a solution of the poison. Thus the soluble salts of cadmium yield, with sulphuretted hydrogen, a rich yellow precipitate resembling closely that produced by arsenic, and this also gives a metallic sublimate when heated with soda-flux. There are, however, these striking differences: the yellow compound of arsenic is soluble in ammonia, that of cadmium is insoluble,—the compound of arsenic is insoluble in strong hydrochloric acid, that of cadmium is perfectly soluble. Of the dried precipitates, the sulphuret of arsenic is not perceptibly affected in the cold by strong hydrochloric acid, that of cadmium is dissolved readily in the cold with the evolution of sulphuretted hydrogen gas; and a colorless salt of cadmium (chloride) is thereby formed, precipitable as a white carbonate by alkaline carbonates. A solution of a salt of cadmium is immediately precipitated, of a rich yellow color, by hydrosulphuret of ammonia,—that of arsenic is *not* precipitated by this agent. There are many other differences: thus cadmium, when boiled with diluted muriatic acid, is not precipitated on copper under the same circumstances as arsenic, and it does not combine with hydrogen to form a combustible gas. An objection, on the ground of the strong similarity of cadmium to arsenic, was unsuccessfully taken to the chemical evidence given on the trial of Mrs. Burdock at Bristol, in 1835. 2. *Tin.* A persalt of tin is precipitated of a dusky yellow color by the gas; but the precipitate is destitute of all the properties of sulphuret of arsenic: it is insoluble in ammonia, and it gives no metallic sublimate when heated with flux. A solution of tin is also known from one of arsenic, by its being instantly precipitated by the hydrosulphuret of ammonia. 3. *Antimony.* A solution of this metal is precipitated of a rich orange-red (not yellow) color by the gas,—the precipitate yields no metallic sublimate with flux, and the solution of antimony is also precipitated by hydrosulphuret of ammonia. If sulphuret of antimony should be mixed with sulphuret of arsenic, the antimony is retained and the metallic arsenic alone volatilized by the process under Exp. 8 (p. 352). 4. *Uranium.* A solution of a persalt of uranium gives, with a current of sulphuretted hydrogen gas, a yellow-brown precipitate, wholly unlike that caused by arsenic. This precipitate differs from that of sulphuret of arsenic, in being insoluble in ammonia, soluble in hydrochloric acid, and in yielding no metallic sublimate with soda flux. Besides, a solution of a uranium salt is precipitated by hydrosulphuret of ammonia.

MARSH'S PROCESS. HYDROGEN TEST.—This process for the detection of arsenic was first proposed by Mr. Marsh, of Woolwich, in the year 1836. It is based on the decomposition of arsenious acid and the soluble compounds of arsenic, by hydrogen evolved in the nascent state from the action of diluted sulphuric or hydrochloric acid on zinc. The apparatus is of the most simple kind, and is so well known, as to need no description. It has undergone various modifications, and has received the names of various supposed inventors or improvers, but the principle is the same, and the sole merit of the invention must be assigned to Mr. Marsh. The arsenic may be introduced into the short leg of the tube in the state of powder; but it is better to dissolve it in water, by boiling, either with or without the addition of a few drops of a solution of potash. The metallic arsenic combines with a portion of the hydrogen, forming *arsenuretted hydrogen gas*, which possesses the following properties: 1. It has an offensive odor, somewhat similar to that of garlic. 2. It immediately blackens a solution of nitrate of silver. This may be proved by holding a piece of filtering paper, wetted with a solution of nitrate of silver, in the current of gas as it escapes from the jet. The silver is reduced, and presents a black metallic appearance. If the gas is passed into a tube containing a weak neutral solution of nitrate of silver, a black precipitate of reduced silver is formed, and the liquid, on evaporation, yields arsenic acid or red arseniate of silver. 3. It burns with a pale bluish-white flame, and evolves, during combustion, a white smoke (arsenious acid). 4. A cold plate of glass or white porcelain, held in the flame near the point, receives a dark stain from the deposit of arsenic upon it. This stain is composed in the centre of pure metallic arsenic, which may be sometimes raised up in a distinctly bright leaf of metal,—immediately on the outside of this, is an opaque black ring (suboxide of arsenic?), which, when viewed by transmitted light, is of a clear hair-brown color at the extreme edge: if the quantity of arsenic be very small, the metallic lustre and opacity may be wanting, and the deposit will be merely a film of a brown color by transmitted light. On the outside of this black ring is a thin wide film, of a milk-white appearance, which is nothing more than arsenious acid reproduced by the combustion of the gas. 5. A white saucer, or a slip of card or paper moistened with ammonio-nitrate of silver, held about an inch above the point of the flame, will be found, if arsenic be present, to be colored yellow, from the reproduced arsenious acid being absorbed, and forming yellow arsenite of silver, easily soluble in ammonia. If a current of the gas be conducted through a tube of hard German glass, and a spirit-lamp applied to the tube during the passage of the current, metallic arsenic of a steel-gray color will be deposited on the glass at a short distance from the flame. This may be removed, and tested by nitric acid. (Exp. 7, ante, p. 348.)

This process is probably the most delicate of all these which have been devised for the detection of arsenic; but for this reason it requires the greatest caution in its application. Its delicacy had been sometimes improperly estimated by the assumed weight of the metallic deposit on glass; whereas it is certain that the quantity of arsenic in one infinitesimal deposit, if transferred to the apparatus, would give no indication whatever of the presence of arsenic. In operating on the poison it must be remembered that, by this process, we are dividing and subdividing the metal into a series of deposits, the weight of some of which might not be equal to the millionth part of the weight of the arsenic which is actually furnishing them. More or less arsenic is always lost during the combustion of the gas; and most of the apparatuses are so constructed, that they allow of the escape of this poisonous gas; a fact which may be demonstrated by placing a solution of nitrate of silver on filtering paper over the open end of the tube. *Objections.*—Other substances will combine with nascent hydrogen, and when the gas is burnt, a deposit will be formed on glass or porcelain, which may be mistaken for arsenic. A liquid containing antimony, selenium, phosphorus, sulphur, or even some kinds of organic matter, may produce a compound with hydrogen, which, when burnt, will leave a dark deposit or stain on glass. The only objection of any practical force is that founded on the presence of *antimony*, which, as a result of medicinal use, may be present in the liquids as well as in the tissues of a dead body. A current of antimonuretted hydrogen gas reduces silver from a solution of the nitrate, but it has not the odor of arsenuretted hydrogen. It burns with a very pale lemon-yellow flame, and forms a white smoke (oxide of antimony). When a solution of ammonio-nitrate of silver, in a saucer, is exposed to this white vapor, there is a black stain, in place of the yellow deposit produced by arsenic. The differences between the arsenical and antimonial deposits obtained by the process of Marsh are, however, well marked. The antimonial deposit has rarely a bright metallic lustre, except when seen on the reverse side of the glass. By transmitted light, the deposit is of smoky-black color, while that of arsenic is hair-brown. Numerous suggestions have been made for distinguishing a deposit of arsenic from that of antimony. The plan which I find to be best adapted for this purpose has been already described in reference to arsenious acid and reduced arsenic. (Exp. 7, ante, page 348.) Receive the deposit from the burning gas on the interior of a small white porcelain capsule. Add a few drops of strong nitric acid. The deposit will be immediately dissolved. Evaporate gently to dryness. Moisten the dry residue with one or two drops of water, and then add a few drops of a *strong* solution of nitrate of silver. If the stain was owing to arsenic wholly, or in part, a brick-red colored precipitate will immediately appear. This will be more or less distinct, according to the quantity of arsenic present. The red precipitate (if owing to arsenic) is entirely soluble in ammonia. A deposit of antimony thus treated, leaves a white residue (oxide of antimony), insoluble in water. Nitrate of silver added to it produces no colored precipitate; but if a little ammonia be brought near, either in vapor or liquid, and a solution of potash is added, a precipitate is formed, which becomes black by standing. Hydrosulphuret of ammonia dissolves the antimonial deposit immediately, and on evaporation leaves an orange-reddish colored film of sulphuret of antimony, soluble in hydrochloric acid, and insoluble in ammonia. The hydrosulphuret does not readily dissolve the arsenical deposit, but when gently evaporated, it leaves a bright-yellow film (sulphuret of arsenic), not soluble in hydrochloric acid, but soluble in ammonia. Imponderable quantities of the two metals may be thus easily identified. In testing these minute films for arsenic, hydrochloric acid must not be used with the nitric, since, on evaporation, a portion, or the whole of the arsenic may be volatilized, and lost as chloride of arsenic.

It will be observed, in the production of antimonuretted hydrogen, that antimony is rapidly deposited in the form of a fine black precipitate in the tube. This arises from the chemical action of the zinc. The antimonuretted hydrogen cannot be kept long, as the antimony is speedily separated from it. Arsenuretted hydrogen may be kept for use for a much longer period, but, sooner or later, some arsenic is deposited on the zinc, as well as on the metallic parts of the apparatus. Stopcocks are thus coated with a film of arsenic; hence fresh zinc and a clean stopcock should be used on each occasion, or the arsenic of a former operation might be erroneously referred to a substance under examination. The operator must not only look to the state of the apparatus, but he should also satisfy himself of the absolute purity of his materials.

The zinc, sulphuric, and hydrochloric acids, which are employed in Marsh's process, are often contaminated with arsenic. Dr. Clark, of Aberdeen, informed me, some years since, that he had not discovered a specimen of zinc free from arsenic, when about an ounce of the metal was used in an experiment, and the hydrogen gas evolved was tested by a solution of nitrate of silver: but pure distilled zinc may now be procured from respectable druggists. The sulphuric acid of commerce sometimes contains a large quantity of arsenic. The late Mr. Scanlan found that 2000 grains of one specimen of acid yielded 1·5 grains of sesquisulphuret of arsenic. (Pharm. Jour. Aug. 1844.) From the great demand for a cheap and impure sulphuric acid for agricultural purposes, it is now the exception to the rule to find this acid free from arsenic. Hydrogen procured by this acid is often contaminated with arsenic to a dangerous extent. An impure sample of acid led to the death of an Irish chemist, who breathed hydrogen thus produced, and who had neglected to test the acid before use. The impurity of this acid leads to the contamination of hydrochloric or muriatic acid with arsenic, for the cheap arsenical acid is now largely employed in this manufacture. The best answer to all objections based on the presence of arsenic from accidental sources is, that the materials were tried repeatedly before the suspected liquid was introduced into the apparatus. If no sublimate or deposit be formed until *after* the introduction of the suspected liquid, it is evident that the arsenic must have been in the liquid introduced; a fact which may be considered as clearly established, if, on removing the liquid and washing out the tube, no deposits whatever result from employing the portions of the same acid and zinc.

These are, I believe, the only tangible objections to the use of Marsh's test, and they are not difficult of removal, when ordinary care is taken. It will be apparent, that not one of these objections could apply, except to those cases where Marsh's test is relied on as the *sole* and exclusive chemical proof of the presence of arsenic; but in most instances, when this process is *safely* applicable, other tests are also applicable; and it does not at all diminish the merit of this most useful and ingenious invention, to say that the results which it furnishes should be corroborated by the use of some of the other tests, if it were only for the sake of preventing any plausible objections to the inference derivable from its employment. The great object of chemical evidence is not to show a court of law what may be done by the use of *one* test only, by peculiar manipulations on imponderable traces, but to render the proof of the presence of poison in the substance examined most clear and convincing. If, in any case, we have no other evidence to offer than that furnished by Marsh's process,—a case in which the quantity of poison must be infinitesimal, and the metallic deposits proportionally minute,—it would be better to abandon the evidence altogether, than to maintain that poison is present from results which admit of no sort of corroboration; for all who have experimented on the subject, must have perceived the utter inefficacy of applying liquid tests to determine the chemical properties of imponderable and scarcely visible sublimates. This appears to me to have been the most objectionable part of the evidence in the well-known case of Madame Laffarge (1840). Orfila admitted that he had obtained only a few deposits, so slight that they could not be weighed. He estimated the united weight at half a milligramme (·0077 gr.), or about the one hundred and thirtieth part of a grain.

When reliance is placed on the blackening of a solution of nitrate of silver as evidence of the presence of arsenic, it must be remembered that sulphuretted hydrogen will produce the same effect, and that sulphur is often contained in zinc. The gas should either be first passed through a solution of a salt of lead,—or so entirely decomposed by heat, that metallic arsenic is obtained in the tube through which the current part is passing.

Delicacy of Marsh's process.—Marsh's process is undoubtedly one of great delicacy. MM. Danger and Flandin assert that metallic deposits may be procured when the arsenic forms only the 2,000,000th part of the liquid examined. (De l'Arsenic, 83.) M. Signoret states that he has procured metallic deposits with only the 200,000,000th part of arsenic in the liquid: this is in the proportion of one grain of arsenic dissolved in about 400,000 ounces, or 3000 gallons of water! As the delicacy of this test has been already made a subject of discussion in a court of law (The Queen against Hunter, Liverpool Spring Assizes, 1843), it may be proper to offer a few remarks respecting it. It was stated at that trial, that the one-millionth part of a grain of arsenic might be rendered visible by Marsh's test; and the judge, guided by this statement, put the question to another medical witness, whether arsenic could be so removed from the stomach in three days, as that it would be impossible to discover the *one-millionth part of a grain in the body.* It appears to me that the facts relative to the delicacy of tests are not always stated with sufficient clearness on these occasions. Thus we have to consider two points: 1. The total quantity of poison experimented on; and 2. the degree of dilution, or the total quantity of liquid in which the poison is dissolved or suspended. There is no doubt that considerably less than the millionth part of a grain of arsenic may, by Marsh's test, be rendered *visible* on a glass plate: it is possible to distinguish with the eye a piece of leaf-gold which would weigh less than the ten-millionth part of a grain; but the practical question is, whether this test will enable us to discover arsenic in a single drop of a solution, made by dissolving one grain of the poison in a million grains or sixteen gallons of water! If not, the statement amounts to nothing; for it is clear that if more than one drop of such an extremely diluted solution be taken, the test is acting upon a larger quantity of arsenic than the above form of expression would indicate. I have generally found that the fractional quantity stated to be detected, referred rather to the degree of *dilution* than to the *absolute quantity of poison* present: whereas a test may fail to act, as it has been already stated, either from the smallness of the quantity of poison present, or from the large quantity of water in which it is diffused. The results of my own experiments are, that when arsenic is mixed with the acid liquid in a tube capable of holding two fluid-ounces, very faint and scarcely perceptible deposits begin to be formed on a glass plate, with a quantity equal to the 2160th part of a grain: the diffusion here being equal to two million times the weight of the poison. With the 1080th part of a grain in the same quantity of water (the arsenic forming therefore one-millionth part), slight brown annular stains were procured. The annular form is probably due to the central portion of the minute film being volatilized by the heat of the point of the flame: unless the glass is speedily removed, the whole of the deposit may vanish. With the 720th of a grain, the arsenic being in the proportion of about the 800,000th part of the liquid, the stains were much more decided, but quite imponderable. With the 100th grain in one fluid-ounce of water (the 48,000th part), and the 67th grain in two fluid-ounces (the 64,800th part), the deposits on glass were decided and characteristic; and it is at this point that the process begins to be safely available for the purposes of legal medicine. M. Villain has attempted to determine how many metallic deposits can be obtained from a given weight of arsenious acid. The result at which he arrived is, that 1-65th part of a grain of white arsenic will yield on an average 226 metallic deposits of an average diameter of the 1-12th of an inch. (Jour. de Chimie, 1846, p. 611.) The average weight of each, therefore, even supposing there were no loss, would be about the 1-15,000th part of a grain.

Reinsch's process.—Hugo Reinsch first published an account of this process, which originated in an accidental discovery of arsenic in muriatic acid, in 1843. (See Ann. d'Hyg. 1843, vol. i, p. 489.) Soon after the announcement, I examined the application of the process to the purposes of Medical Jurisprudence, and a full account of the results was published in the British and Foreign Medical Review, for July, 1843, p. 275. It has since been extensively employed in this country in medico-legal practice. While it is open to fewer objections than the process of Marsh, it is preferable in its simplicity, and in the facility of its application. It enables the analyst to trace arsenic to a minute degree in all its combinations, if we except arsenic acid and the arseniates; and in reference to these compounds, it is inferior in delicacy to the process of Marsh. One substance only is required of which the purity must be guaranteed, namely, the muriatic or hydrochloric acid. (Exp. 5, p. 347, ante.) Metallic copper, either in the form of freshly polished foil or wire, is used for the separation of the metal. The material best adapted for this process is the finest copper-wire woven into a gauze containing from twelve to sixteen thousand apertures to the square inch. A small piece of this, by reason of the extensive surface presented, will enable the analyst to collect a comparatively larger proportion of arsenic than would be deposited on the foil. The arsenic adheres to it with greater firmness, and the gauze will indicate by a change of color the presence of the poison, when the appearance on the foil would be indistinct.

The liquid suspected to contain arsenic is mixed with from one-sixth to one-eighth part of its volume of muriatic acid (free from arsenic), and brought to the boiling-point. A solid is simply boiled in distilled water containing about the same proportion of acid. When brought to the boiling-point, about half a square inch of copper gauze is introduced, and if arsenic is present, even in a small quantity, this is indicated by the copper acquiring an iron-gray color. The gauze is removed, washed in water to free it from any trace of acid,—dried on blotting paper and in a warm current of air, rolled into a small cylinder and placed in a dry and warm reduction-tube. Heat is now gradually applied to the cylinder of coated copper, and the metallic arsenic, in subliming, is deposited in a cool part of the tube in the form of a ring of brilliant octahedral crystals of white arsenic. These may by identified by the microscope, and then tested in the manner described (under Exp. 7, p. 348 or p. 349). When the quantity of arsenic is small, the polished copper merely acquires a faint greenish-blue or bluish tint, and the time required for the deposit is materially affected by the quantity of water present, or in other words, the degree of dilution. But one great advantage is, that we are not obliged to dilute the liquid in the experiment, and there is no material loss of arsenic after the copper is introduced, as in Marsh's process; the whole may be removed and collected by the introduction of successive portions of the metal. This process is extremely delicate, the results are very speedily obtained, and are highly satisfactory. Among the cautions to be observed are these: 1. Not to employ too large a surface of copper in the first instance; and 2. Not to remove the copper from the liquid too soon. When the arsenic is in small quantity, and the liquid is much diluted, the deposit does not take place sometimes for half an hour. If the copper is boiled in the liquid for an hour or longer, it may acquire a dingy tarnish, in the absence of arsenic, from the action of the acid only (oxychloride of copper). This is known by its yielding no crystalline sublimate when heated, its want of metallic lustre, as well as by its being easily removed by friction.

Objections.—Certain objections have been urged to this process. 1. Arsenic may be present in the muriatic acid: this is at once answered by boiling the copper in a mixture of the muriatic acid and water before adding the suspected liquid. This should always be a preliminary experiment. In the case of Mrs. Wooler (Durham Winter Assizes, 1855), some doubt was thrown on the scientific evidence by reason of the use of arsenical muriatic acid. The discovery of the impurity was not made, until after the analysis was completed. 2. Another objection is, that other metals are liable to be deposited on copper under similar circumstances. This is the case with *antimony*, whether in the state of chloride, or of tartar emetic; and it is not always easy to distinguish by the appearance the antimonial from the arsenical deposit. Should the quantity of antimony be small, the deposit is of a violet tint: if large, of an iron-gray color, resembling arsenic. In this case, a portion of the solution may be greatly diluted, when the peculiar violet-red color of the antimonial deposit will be made apparent. There is one answer to these objections, namely, that from an *arsenical* deposit, octahedral crystals of arsenious acid may be procured by *slowly* heating the copper, and the crystals may be proved by other tests to be those of white arsenic. Such a corroboration is necessary, because the crystalline form of arsenious acid is not always distinguishable by the eye; and the antimonial deposit gives a white amorphous sublimate, which, however, is quite insoluble in water. Care must be taken not to mistake minute spherules of water, mercury, or muriatic acid for detached crystals of arsenious acid; and here the microscope will be found of great service. The facility of applying Reinsch's process renders it necessary that the experimentalist should be guarded in his inferences. It is not merely by the production of a deposit on copper that he judges of the presence of arsenic: but by the conversion of this deposit to arsenious acid, demonstrable by its crystalline form and its chemical properties. If a deposit take place on copper, and arsenious acid cannot be obtained by heating it, then the evidence of its having been caused by arsenic is insufficient. Owing to the neglect of these corroborative results, antimony and other substances have been occasionally mistaken for arsenic. Bismuth, silver, mercury, gold, and platina, are deposited on copper from diluted solutions under similar circumstances: the four last-mentioned metals being rapidly deposited in a *cold* acidulated solution. The deposits of the three first metals have a silvery white appearance, quite unlike that of arsenic, and the only volatile metal among them is mercury, which is sublimed in the form of bright metallic globules, visible to the naked eye, or by the aid of a lens or microscope. Gold produces a yellowish or bronze-colored deposit. Platina produces a deposit like arsenic, but it is fixed: no sublimate can be obtained from it. An alkaline sulphuret, or sulphuretted hydrogen, if present, as in a putrescent liquid, may give a tarnish to copper; but this is a fixed stain of sulphuret of copper. All objections are at once answered by the production of a crystalline sublimate, and the chemical proofs of its properties. The above facts show that a mere deposit or tarnish on copper cannot be relied on as a proof of the presence of arsenic.

Delicacy of Reinsch's process.—This test failed to detect the 4000th part of a grain of arsenic in thirty drops of water, the dilution being equal to 120,000 times the weight of the arsenic. The deposit on copper commenced with a purplish-colored film, when the quantity of arsenious acid was equal to the 3000th part of a grain in thirty drops of water, or under a dilution of 90,000 times its weight. It was very decided with the 2000th part of a grain in the same quantity of water, but in neither of these cases could octahedral crystals of arsenious acid be obtained by heating the copper. The following experiments will show how this test is liable to be affected by dilution: The copper was coated in a few seconds, when boiled in a solution containing the 4000th part of a grain in ten drops of water, although the test had failed to detect the same weight of arsenic in three times that quantity of water. So, again, the 2160th part of a grain in thirty drops of water gave an arsenical deposit on copper; while the same quantity in half an ounce of water, did not produce any change of color in the metal. By concentrating a diluted solution with the copper immersed in it, we shall, sooner or later, secure a deposit of the arsenic and at the same time prevent it from escaping in vapor as chloride of arsenic. The deposit adheres with great firmness to the copper gauze. In examining one of these deposits after it had been loosely wrapped in paper for a period of nearly *fourteen years*, I found it of a brownish-black color and iridescent: the eighth of a square inch, when heated, still gave a well-marked ring of octahedral crystals of arsenious acid.

There can be no doubt that Marsh's process will enable an operator to detect a smaller quantity of arsenic than that of Reinsch; but when Reinsch's process fails to detect arsenic, it would be hardly safe to trust to the evidence furnished by the process of Marsh alone. Our law authorities have not yet been inclined to receive with favor chemical evidence when it is based on the fiftieth or the one hundred thousandth part of a grain, and the numerous mistakes respecting normal and cemetery arsenic, which have owed their origin to too great a reliance on Marsh's process, are a justification of this distrust.

Arsenic in liquids containing organic matter.—Arsenious acid, when in a state of solution, is not liable to be precipitated by any animal or vegetable principles, although all such substances render it less soluble in water. The liquid for analysis should be filtered through muslin, cotton, or paper, in order to separate any insoluble matters. Should it be colored, this is of little moment, provided it be clear. If viscid, it should be diluted with water and boiled with a small quantity of muriatic acid; on standing, a deposit may take place, and this should be separated by a filter. A portion of the original liquid before boiling should always be filtered and tested for arsenic, in order to determine whether any of the poison is present in a state of solution.

As a trial-test, we may boil in a portion of the liquid, strongly acidulated with pure muriatic acid, a small piece of copper gauze fixed to the end of a fine and polished copper wire. In a few seconds, if arsenic be present, the copper will acquire a gray metallic coating. If, after half an hour, the copper remain unchanged, the arsenic, if present, must be in extremely minute proportion; if, on the other hand, the copper be covered by a gray deposit, it should be dried and heated in a reduction-tube in the manner already described. From several such slips of copper, or copper gauze, a quantity of metallic arsenic may be procured, sufficient, on reconversion to arsenious acid, to allow either of a solution in water being made, to which all the liquid tests may be applied, or of its conversion by nitric acid to arsenic acid. (Exp. 7, ante, p. 348.) Some organic matter may be deposited in the meshes of the gauze; this may be removed, after washing in water, by warming the gauze in alcohol, or immersing it in ether or chloroform; it should be again washed in water and dried before heat is applied to it. When much oily matter is present, it is better to boil the organic substance with diluted muriatic acid, strain the liquid from it, through calico, and filter it through *a wet filter* before introducing the copper gauze. An even coating of arsenic was by this process obtained on copper gauze from the decomposed tissue of the stomach of a person who had been buried nearly two years. As the gauze is remarkably hygrometric, it always requires to be thoroughly dried in a warm current of air before it is submitted to heat in a reduction-tube. The arsenical nature of the deposit on copper must be then established by the microscope and the tests already described (ante, p. 358). In a fine sublimate, derived from some hay in the stomach of a horse that had been killed by arsenic, I counted twenty-eight distinct crystals of arsenic (arsenious acid), in a space the 1-2000th of an inch square. The greater number of these crystals had a diameter of the 1-2000th of an inch; some distinct octahedra, which had a diameter less than the 1-4000th of an inch, were recognized among them.

By this process, the 144th part of a grain of arsenic was detected in two fluid-drachms of gruel, milk, porter, and other organic liquids, in so many different experiments. It was also thus easily separated from wine, brandy, the liquid contents of the stomach, the blood, and the tissues of the viscera. (Brit. and For. Med. Rev. July, 1843, p. 281.) Here our analysis might be closed, if the object were to determine only the *presence* of arsenic, since a case can rarely occur in medico-legal practice, in which it would be necessary to extract the *whole* of the poison from the contents of the stomach or intestines, or from all parts of the body.

Another process for procuring evidence of the presence of this poison in liquids, consists in transforming the arsenious acid to the state of sulphuret, by a current of sulphuretted hydrogen (ante, p. 351). The liquid should be first boiled, a little diluted muriatic acid added, and then filtered to separate organic matters. This object may be further accomplished by adding to the filtered liquid, when cold, one-third of its bulk of alcohol, again filtering and concentrating the liquid by evaporation. Sulphuretted hydrogen gas may now be freely passed into the liquid acidulated with either of the acids mentioned. When the precipitation has ceased, the liquid should be filtered, the precipitate collected, and dissolved in ammonia to separate it from organic matter; it may then be obtained by evaporating the ammonia. That the yellow compound is sulphuret of arsenic may now be proved by the tests elsewhere described (ante, p. 351). The sulphur process succeeds only when the quantity of arsenic is large.

Arsenic is soluble in oil. It may require analysis either in this state, or mechanically diffused in fat, butter, tallow, or similar substances. From these mixtures it may be separated by boiling them in a sufficient quantity of water, with about one-tenth part of muriatic acid. The aqueous solution may then be freed from the oil or fat by passing it through a filter previously saturated with water, and the arsenic obtained by sulphuretted hydrogen, or by Reinsch's process. (See ante, p. 358.)

Contents of the stomach—Vomited matters.—The contents of the stomach are often mixed with lumps of arsenic, which may be separated by throwing those portions that do not pass through a filter into a large glass of distilled water, and after giving to it a circular motion, suddenly pouring off the supernatant liquid, when the heavy portions containing arsenic will be found at the bottom. The lumps may sometimes be left in the contents; they may then be easily removed, dried on filtering paper, and tested (see ante, p. 347). If the arsenic has been taken in fine powder, there will be no lumps, but it will probably be deposited in masses, mixed with mucus and blood, on the coats of the organ in those parts where it is much inflamed and ulcerated. The arsenic in this state looks like moistened plaster of Paris, but it is of a darker color, and when examined by a lens it is crystalline. It may be removed on a spatula, spread in masses on filtering paper, and slowly dried. As it dries, the granules will detach themselves from the mass. It is necessary to examine the arsenic carefully, with a view of determining whether the white particles are or are not mixed with either of the coloring matters required by the Act of Parliament (ante, p. 320),—indigo or soot. The microscope should here be used for the examination of the dried sediment, and after this examination it may be tested either by the reduction test (p. 348) or by Reinsch's process (p. 358). The suspected particles, or even the stained portions of paper on which the organic matter has become dried, may be boiled with muriatic acid and copper gauze. Mucus, blood, or even a layer of the mucous membrane of the stomach may be thus easily tested. This is in general the only method which it is now necessary to employ. By the use of numerous tests and processes, a witness exposes his evidence most unnecessarily to many ingenious objections. It is sufficient to obtain the deposit on copper; to convert this by heat to crystallized arsenious acid, and to test the sublimate. We thus avoid the troublesome and complex method of separating organic matter from arsenic. Care must be taken in examining a stomach not to confound pieces of bread, or lumps of fat or adipocere with arsenic. Small portions of such substances appear very much like lumps of the poison. In the stomachs of exhumed bodies a crystalline substance is found, resembling arsenic in appearance, but not in properties. It is the ammonio-phosphate of magnesia derived from putrefaction.

The liquid contents may yield no arsenic, although the poison is present. I have found solid arsenic spread over the coats of the stomach in two cases, when the liquid contents yielded no traces of the poison in solution. In the same way I have detected no arsenic dissolved in tea when it was abundant in the sediment. (Queen v. Lever, 1845.) If none should be found either dissolved in the contents of the stomach or on the surface of the organ, we must remove the inflamed and ulcerated portions of the mucous coat, or even the whole of the stomach cut into small pieces, and boil the cut portions with diluted muriatic acid and copper for half an hour. The liquid may be then filtered and tested. It may happen that no arsenic will be detected in the contents of the stomach or vomited matters, until after they have been boiled for at least one or two hours, and the organic matter broken up.

Detection of absorbed arsenic in the tissues.—When arsenic cannot be detected in the liquid contents of the stomach, it is necessary to examine the blood, secretions, muscles, or viscera of the deceased for that portion of the poison which has been *absorbed.* In most cases of acute poisoning, arsenic will be found, but in variable quantities, in every one of the soft structures of the body—more abundantly in the organs of the abdomen than elsewhere. In general a medical witness has it in his power to make a selection; but even here criminal ingenuity may be exerted to defeat his evidence. In a case tried in France, in 1846, the body of a man named Glœckler, who was alleged to have died from poison administered by his wife, had been clandestinely removed, and thrown into the soil of a privy, where it was subsequently found. The abdomen had been opened, and the organs removed, with the intention of obliterating all traces of the criminal act which had been perpetrated. The proofs of criminality rested entirely upon the chemical evidence, for the symptoms were not well marked. It was clearly shown at the trial, that the wound in the abdomen had been made after death; and arsenic was readily extracted from the soft parts of the body in sufficient quantity to be weighed. The accused was convicted. (Gaz. Méd. Sept. 12, 1846, 726.) From this account, it will be perceived, that but for the process of detecting arsenic in the tissues, this crime must have passed undetected and unpunished. Except by the entire destruction of the body in a case of arsenical poisoning, a criminal cannot now defeat the objects of a chemical investigation. Dr. Schaffer met with a case in which an attempt was made by the accused to destroy the dead body by fire. A woman had suffered from symptoms of poisoning by arsenic, and died in about eight hours. Notice was given to the husband that there would be an inspection of the body, and on the next night his house was found in flames. The dead body of the wife was so burnt that it could scarcely be recognized. The stomach, however, had not been entirely destroyed, and in the shrivelled remains of this, some particles of solid arsenic were found. The facts were clearly proved against the husband. (Casper, Vierteljahrschrift, B. 14, H. i, July, 1858, p. 88.)

In the case of the Queen v. Hunter, tried at the Liverpool Spring Assizes, 1843, arsenic could not be detected in the contents of the stomach and bowels; as no analysis was made for absorbed arsenic (ante, p. 50), the benefit of the omission was given to the accused person. In the case of the Queen v. Thomas (Cardiff Summer Assizes, 1843), no arsenic was found in the contents of the stomach and intestines of one of the deceased persons; but the poison is stated to have been detected in the liver by the coarse process of incineration with nitre. This evidence, although attacked in cross-examination on the ground (now refuted) that arsenic was a natural constituent of the body, was received as a satisfactory proof of the presence of the poison. There are few cases in which this branch of the analysis should not be resorted to, although it entails much additional trouble. The detection of arsenic in the *tissues* makes it clear, under the limitations elsewhere pointed out (ante, p. 69), that the poison must have been introduced during life, and that it has most probably caused death; its detection merely in the *contents* of the stomach or intestines, does not give this absolute proof. Besides, the poison found in these contents is not that which has caused death; this, as it has been elsewhere stated, must be referred to the portion of poison which has entered the blood by absorption. It is worthy of remark that under the Medical Witnesses' Act (6 and 7 William IV, c. lxxxix), a medical practitioner is only required to make "an analysis of the *contents* of the stomach or intestines," and for this only is a fee allowed!

The processes commonly employed for the discovery of arsenic in the tissues, are those of Marsh and Reinsch; and *Marsh's process* has been almost universally employed by continental and English chemists. When an organic liquid, containing arsenic, is placed in the apparatus, there is a frothiness produced which interferes with the combustion of the arsenuretted hydrogen. Various plans have been proposed to remove this inconvenience by destroying the organic matter, and procuring the arsenic in a form convenient for testing. In 1839, Orfila suggested the deflagration of the organic matter (brought to a state of dryness) with powdered nitre. He subsequently advised that the organic matter, finely cut up, should be boiled in a weak solution of potash, and mixed with twice its weight of pure powdered nitre. The saline residue obtained on evaporation was then deflagrated by projection in small quantities in a red hot crucible. Another of his plans consisted in decomposing the organic matter by strong nitric acid,—bringing it to the state of a dry carbonaceous mass,—and acting on this by nitro-muriatic acid. The arsenic was subsequently dissolved by water, and then placed in the apparatus. (See his Toxicologie, 1852, i, p. 493.) These processes occasioned a great loss of arsenic, and they had the disadvantages that the presence of any nitric acid or a nitrate interfered with the production of arsenuretted hydrogen. MM. Danger and Flandin recommended the complete carbonization of the organic matter by heating it in a quantity of strong sulphuric acid (proved to be free from arsenic), equal to about one-third of the weight. It thus forms a tarry-looking mixture, which should be brought to dryness. The dry carbonaceous mass is then treated on the plan recommended by Orfila. (Flandin, Traité des Poisons, i, p. 618.) The vapors evolved during this operation are of the most offensive and persistent description.

If sulphuric acid can be obtained pure, there is no doubt that this is the best mode of carbonizing organic matter. The carbonaceous ash should not be too strongly heated, or, as pointed out by Blondlot, there will be a loss of arsenic. (Comptes Rendus, 1845, ii, 32.) It will be found better for the conversion of the arsenious into arsenic acid, to use strong nitric in preference to nitro-muriatic acid, as this will avoid the volatilization of arsenic as chloride. After heating the mixture to expel the greater part of the nitric acid, the ash may be drenched with water until all that is soluble is removed. This may be brought to dryness in a porcelain vessel, and again treated with nitric acid several times, until the residue is without color. The acid residue dissolved in water should be neutralized by pure carbonate of potash, and when again brought to dryness, the arseniate of potash thus produced (if arsenic were present) may be separated from the other salts by a small quantity of water. This solution may be introduced into Marsh's apparatus. If a deposit of metal is obtained on glass, or porcelain, or a ring of metal in a tube, by heating the current of gas, these must be tested by the methods already suggested (ante, p. 348). When Marsh's process is employed, I have found this to be the best plan of proceeding for destroying organic matters and avoiding a loss of arsenic.

In the event of Reinsch's process being selected at this stage, it will be necessary to reconvert the arsenic acid, obtained by the above-mentioned carbonizing process, to arsenious acid. This is effected by evaporating it to dryness with a strong solution of sulphurous acid.

Fresenius and Babo destroy the organic matter by hydrochloric acid and chlorate of potash, and advise a series of proceedings of a most minute and elaborate kind. In fact, this mode of detecting arsenic may be designated an exhaustive process. It provides for the exclusion of lead, bismuth, mercury, copper, tin, antimony, and other metals; but in thus excluding many bodies which are never likely to be found, it encumbers the investigation with the employment of so many chemicals, that a question might fairly arise whether arsenic had not been actually introduced into the organic matter during the operation. I have known only one case in which it has been medico-legally employed in this country, that of Reg. v. Wooler (Durham Winter Assizes, 1855), and there fortunately the proof of death from arsenic was so clearly made out from other facts, that it was unnecessary to make this elaborate mode of testing a subject of cross-examination. The reader who is curious about this process, the complication of which, according to Orfila, surpasses all credibility, will find the details in Orfila (Toxicologie, i, 515, Otto, Ausmittelung der Gifte, p. 34, 1856, and Böcker, Die Vergiftungen, 1857, p. 88).

Reinsch's process.—I have found that *Reinsch's process*, without any preliminary carbonisation, is well adapted for the separation of absorbed arsenic. I have by it separated arsenic from all the organs of the body, excepting the brain and spinal marrow. The organ suspected to contain deposited arsenic, is cut into the thinnest pieces. It is then digested at a gentle heat, with a mixture of one part by measure of muriatic acid (proved to be free from arsenic), and eight parts of water. When the structure of the organ is broken up, a piece of copper gauze, at the end of a fine and polished copper wire, is put into the vessel, and the liquid brought to the boiling-point. If no deposit takes place in a few minutes, a little more muriatic acid may be added. If after half an hour, there is no change of color in the wire or gauze, there is probably no arsenic present. The liquid may now be boiled down on the copper, and the metal again washed and examined. The quantity of gauze used must be small, until there is clear evidence of a free deposit, and then a number of pieces may be successively added until the arsenic ceases to be deposited. The pieces of gauze which are coated, should be well washed in water, and if necessary in alcohol, to separate adhering organic matter. They may then be tested for arsenic by the processes already described. The only precaution required here, is that the muriatic acid should be pure. The water, the copper, the acid, and the vessel, may be tested for arsenic before the addition of the substance supposed to contain it.

In January, 1852, six ounces of a thick turbid fluid were taken from the stomach of a dog supposed to have been poisoned. As there was no poison present,—the copper gauze remaining unchanged in color, five drops of a solution of white arsenic, equivalent to (·06) six-hundredths of a grain of the poison, were put into the mixture, and the whole was well stirred. In ten minutes the copper was coated of a steel-blue color, and crystals were obtained from it by heat. The 16th part of a grain of arsenic was here detected, in about forty thousand times its weight of a complex organic liquid.

Sometimes the red color of the copper is seen, owing to the thinness of the arsenical deposit. In all cases it is advisable to dry the gauze, to heat a portion of it in a reduction-tube, and examine the sublimate, if any, by the microscope, before giving an opinion that arsenic is absent.

Either of these processes will enable the analyst to separate arsenic from the tissues. By Marsh's process he can accumulate in a porcelain capsule, in a state convenient for testing, any number of metallic deposits. In reference to Reinsch's process, as soon as the copper is covered by arsenic, the chemical effect ceases: the use of gauze allows the operator to concentrate a large quantity of arsenic in a small space. This yields a ring of crystals sufficient for testing, or it may be made so by heating a number of pieces successively. There can be no doubt that Reinsch's process is the most simple, and the least open to objection in respect to the casual introduction of arsenic, as neither zinc nor sulphuric acid is required. It has also this advantage; while searching for arsenic in the tissues, it enables the operator to discover and separate at the same time both mercury and antimony, without interfering with the search for arsenic.

There is no doubt that a portion of arsenic is lost by either process, and from the researches of Dr. Geoghegan, it appears that but little more than one-half of the arsenic deposited on copper in Reinsch's process can be reobtained by sublimation in the crystalline state (Dublin Quarterly Journal, Feb. 1851), the rest being retained as arsenuret of copper. So in the combustion of arsenuretted hydrogen in Marsh's process, there is an unavoidable loss of arsenic on each occasion that the jet is opened. Either process is, however, so delicate, that even making due allowance for loss of the poison, the arsenic will be easily revealed when forming not more than from the 1-150th to the 200th part of a grain, and this is itself a point of delicacy in an analysis which, when the issues of life and death are involved, might almost suffice to justify a reasonable distrust of the resources of science. Orfila professed to have detected the 2,000,000th of a grain by Marsh's process, but the smallest quantity on which he ever ventured to give evidence before a legal tribunal was in the well-known case of Madame Laffarge. His evidence, however, was strongly objected to. He admitted that the quantity which he obtained from the body of the deceased was too small to be weighed, but estimated it roughly at half a milligramme, *i.e.* about the *one hundred and thirtieth* part of a grain! In this county, I am not aware that chemical evidence of the presence of arsenic in a dead body has ever been based on a smaller quantity than in the case of Margaret Wishart. Dr. Christison did not detect more than the *one-fortieth* part of a grain of arsenic in the coats of the stomach; but this was deemed sufficient chemical evidence, and the prisoner was condemned and executed. (Edin. Med. and Surg. Journ. xxvi, 23.) The smallest quantity on which I have had occasion to give evidence in criminal trials, was from half a grain to a grain, estimated as the quantity actually obtained in crystals from the stomach, intestines, and tissues. (Reg. v. Chesham, Essex Lent Assizes, 1851, and Reg. v. Bacon, Lincoln Autumn Assizes, 1857.) In these two cases, the accused were tried and convicted on the charge of administering poison with intent to murder. The cause of death was not here at issue.

Nevertheless there is a strong prejudice among lawyers that the chemical evidence is defective unless the quantity found is sufficient to cause death. The irrelevancy and the absurdity of such a proposition, in a medical point of view, has been elsewhere pointed out (ante, p. 185). It would be just as reasonable, in a case in which a man has been killed by a discharge of small shot, to insist upon a failure of proof of the cause of death, because only a single pellet has been found on the body. The value of chemical evidence does not depend on the discovery of any particular *quantity* of poison in the stomach,—it is merely necessary that the evidence of its presence should be clear, distinct, conclusive, and satisfactory. At the same time a reasonable objection may be taken to a dogmatic reliance upon the alleged discovery in a dead body of minute fractional portions of a grain; and considering the great liability to fallacy from the accidental presence of arsenic in the articles used, the chemical evidence in the case of Laffarge was of a most unsatisfactory kind, and should have been rejected by the Court. No man with any respect for his character, or for the common sense of a jury, would preliminary carbonization, is well adapted for the separation of absorbed arsenic. I have by it separated arsenic from all the organs of the body, excepting the brain and spinal marrow. The organ suspected to contain deposited arsenic, is cut into the thinnest pieces. It is then digested at a gentle heat, with a mixture of one part by measure of muriatic acid (proved to be free from arsenic), and eight parts of water. When the structure of the organ is broken up, a piece of copper gauze, at the end of a fine and polished copper wire, is put into the vessel, and the liquid brought to the boiling-point. If no deposit takes place in a few minutes, a little more muriatic acid may be added. If after half an hour, there is no change of color in the wire or gauze, there is probably no arsenic present. The liquid may now be boiled down on the copper, and the metal again washed and examined. The quantity of gauze used must be small, until there is clear evidence of a free deposit, and then a number of pieces may be successively added until the arsenic ceases to be deposited. The pieces of gauze which are coated, should be well washed in water, and if necessary in alcohol, to separate adhering organic matter. They may then be tested for arsenic by the processes already described. The only precaution required here, is that the muriatic acid should be pure. The water, the copper, the acid, and the vessel, may be tested for arsenic before the addition of the substance supposed to contain it.

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QUANTITATIVE ANALYSIS.—The quantity of arsenic met with in a free state in the stomach and bowels after death, is subject to great variation. The quantity found has varied from half a grain to two ounces, or 960 grains (see ante, p. 327). The circumstances which affect this quantity have been elsewhere considered (ante, p. 173). In dealing with a liquid article of food or with the contents of a stomach, assuming that the arsenic is dissolved, we pursue the same plan. In some cases, solid arsenic, in lumps or powder, may be separated by washing from the contents. In this case we simply collect it, dry it, and weigh it. A measured portion (one-fourth or one-sixth) of the liquid should be acidulated with diluted muriatic acid, boiled and filtered. A current of sulphuretted hydrogen may now be passed into it, until there is no longer any precipitation. The liquid should be again boiled, and the precipitated sulphuret of arsenic collected by decantation or on a filter, and thoroughly washed. While still moist, it may be dissolved in ammonia, and the ammoniacal liquid filtered into a balanced capsule, from which the ammonia may be driven off by evaporation. The sulphuret of arsenic dried at 212° may now be weighed, and as every *hundred* grains of sulphuret represent *eighty grains* of white arsenic (100 : 80·4), the quantity of the latter may be found sufficiently close for practical purposes in multiplying the weight of the precipitate by 4 and dividing the product by 5.

When we are dealing with the tissues, the quantity of arsenic is generally too small for the application of this method. In all cases, the *deposited* arsenic is in very small proportions (ante, p. 46), rarely exceeding a few grains in an organ like the liver weighing four or five pounds; and, according to Flandin, although this statement is not in accordance with the experience of others, *nine-tenths* of the deposited arsenic are found in the liver, the other tenth being unequally diffused through the other organs. (Op. cit. i, 564.) As a general rule, the liver will be found to contain the largest proportion, and next to this the spleen and kidneys, the heart and muscles containing the smallest proportion. I have sometimes found none in the liver, while it has been present in the other organs, and even in the bile contained in the gall-bladder.

In order to determine the proportion of absorbed arsenic in an organ (*e. g.* the liver), which, under any circumstances, can be done only approximately, we take a weighed quantity (four ounces), slice it and treat it by Reinsch's process (ante, p. 358), separate the whole of the arsenic by copper gauze, and determine or estimate the weight of the sublimates obtained—doubling this weight to allow for the unavoidable loss, and calculating from the quantity found in four ounces, the quantity diffused through the whole of the liver. Another portion of liver may be examined, if necessary, to correct the result. I believe that the quantity thus determined is always below the amount actually present. Some prefer the determination of the quantity from a given weight, by passing the arsenuretted hydrogen, generated in Marsh's apparatus (by the process described, ante, page 353), into a weak solution of neutral nitrate of silver, until there is no longer any blackening. The arsenic is here supposed to be converted entirely to arsenic acid; the surplus silver is cautiously removed by hydrochloric acid, and after filtration and evaporation to dryness, the arsenic is precipitated from the residue dissolved in water by sulphuretted hydrogen. (Orfila, op. cit. i, 173.) Another plan consists in passing the arsenuretted hydrogen through a tube of infusible glass, dipping into a solution of chloride of gold, and when air is expelled, applying heat to the tube. Metallic arsenic is deposited, and any portion of gas which escapes decomposition is collected and dissolved in the solution of gold which it decomposes. (Orfila, i, p. 512.) The quantity thus obtained, is determined by a process similar to that above mentioned. In pursuing these methods, there may be a loss of arsenic in carbonizing and heating to dryness the organic matter with sulphuric acid; a portion of arsenic is liable to be separated and deposited in the apparatus used; and it is not improbable that a portion combines with the reduced silver and gold.

CHAPTER XXIV.

NON-DETECTION OF ARSENIC—PARTIAL OR ENTIRE ELIMINATION OF THE POISON—NORMAL ARSENIC—ARSENIC NOT A CONSTITUENT OF THE HUMAN BODY—ARSENIC IN ARTICLES OF FOOD—IN SEED-CORN—ITS DETECTION IN EXHUMED BODIES AFTER MANY YEARS—INFERENCE OF DEATH FROM POISON IN SUCH CASES—CEMETERY-ARSENIC—BURIAL OF BODIES IN ARSENICAL EARTH—EVIDENCE FROM THE ANALYSIS OF EARTH—PRESERVATIVE EFFECTS OF ARSENIC.

Non-detection of arsenic.—It is an important medico-legal fact, that in many undoubted instances of arsenical poisoning, not a trace of the poison can be found in the stomach or its contents. Several of these cases have occurred within my knowledge. In one, a girl took an ounce of the poison, and died in seventeen hours: there was much vomiting and purging, and the stomach-pump was used,—facts that might sufficiently account for the non-detection of poison in the body. In a second, nearly *two ounces* of arsenic were swallowed, and the person died in eight hours. No arsenic was discovered in the stomach. Even when there has been no vomiting and purging, the poison is not always found, but then the dose is generally small. Thus, in a case referred to me by Mr. Veasy, no arsenic could be detected in the stomach, duodenum, or their contents, although the patient had had neither vomiting nor purging. Reasons have been already assigned for the non-discovery of the poison (see ante, p. 48 and 174).

It is important, in reference to absorbed arsenic in the *tissues*, to observe, that the poison may be found in them even at an early period, when it is either absent or only doubtfully present in other parts. In a case referred to me by Mr. Gell, coroner for Sussex, in May, 1854, the deceased, Burton, died within *four hours* after he had been attacked with symptoms of poisoning by arsenic. Arsenic was found in small quantity in the stomach; but it was also detected in the liver and spleen (ante, p. 46). The precise time at which the poison was taken, could not be determined; but the fact mentioned shows that its deposition in the tissues takes place very rapidly. In the cases of the Atlee family, referred to me by Mr. Carter, coroner for Surrey, in January, 1854, the body of the mother was exhumed after a month. Arsenic was *not* found in the *stomach* or *bowels;* but it was readily detected in a small portion of the *liver*. The poison had probably been taken several days before death. This fact is of considerable importance in relation to a medical opinion of the presence or absence of poison in a dead body. It is very commonly the practice to confine an analysis to the stomach and bowels only; and when no poison is found therein, to report that no poison exists in the body, and to refer death to natural causes. It is clear, however, from the above case, that such an opinion might be erroneous unless the liver or spleen had undergone a chemical examination. In reserving viscera for analysis, a portion of the liver should, therefore, always be examined. It need hardly be observed that, in these analyses, the organs should be separately examined. The detection of arsenic, in many and different parts of the body, often furnishes important information regarding the time at which the poison was taken. The stomach should be examined separately from the intestines; and among the intestines, it is desirable to make distinct analyses of the duodenum and the rectum. The liver, heart, spleen, and kidneys should each be examined separately, if they have not been placed in contact with each other, or in contact with the stomach. When the quantity of poison is small (as indicated by trial-tests), or but small portions of the organs can be obtained, then it may be proper to combine different viscera in one analysis. In an important case, which was the subject of a criminal trial, the stomach, intestines, liver, spleen, and other organs, were, as I am informed, associated in one mass. This gave much less trouble to the operators; but although arsenic was found in small quantity, it was impossible to fix upon the organ which had contained it, or even to state with certainty that it had not been accidentally introduced in the materials employed in the investigation.

If the person has lived *fifteen* or *sixteen days* after having taken the poison, no trace of arsenic may be found in the stomach, bowels, tissues, or in any part of the body. Orfila had expressed this opinion from his experiments on animals; its correctness has been strikingly confirmed by the case of Dr. Alexander, who died in sixteen days from a dose of arsenic taken by mistake in arrow-root. Dr. Geoghegan, who was deputed to make an analysis of the stomach, and other viscera, found no trace of the poison, either absorbed or unabsorbed, in any part of the body which he examined. (See Med. Times and Gazette, April 18, 1857, p. 389 and p. 40; also ante, pp. 49, 51.)

A case occurred in France, which clearly shows the great importance attached to this part of the analysis. A woman was accused of having destroyed her husband by arsenic. The witnesses who had the investigation of the case, detected arsenic in the liquid contents of the stomach in well-marked quantity; but they omitted to extend the analysis to the tissues. The parties who were required to express an opinion of the cause of death from the medical facts, declared that the proofs of death from arsenic were insufficient, because, in order to justify this inference, the poison should be detected in the *tissues* of the body. The counsel in defence contended that the proof of poison had failed, and the jury acquitted the prisoner. (Gaz. Méd. 3 Janvier, 1846 p. 18.) It is very true that in *an unknown* case the presence of arsenic in the *contents* of the stomach may not be an absolute proof of death from this poison, unless there be at the same time some confirmatory evidence from symptoms and appearances; yet when these exist, the omission to examine the tissues cannot be fairly urged as an objection to the medical opinion of death from poison. On these occasions the proof that death was caused by arsenic, must rest upon the evidence from symptoms and appearances, as well as on the moral circumstances of the case. On the other hand, when arsenic is discovered either in a free state in the stomach, or in an absorbed state in the tissues, a presumption may arise, that if not casually introduced during the chemical analysis, its presence may admit of explanation without reference to a criminal charge.

Normal Arsenic.—Arsenic not a constituent of the human body?—In the year 1838, Orfila announced from his experiments that arsenic was a natural constituent of the human body, existing especially in the bones, and also probably in the muscular system. (Ann. d'Hyg. 1839, ii. p. 467.) This statement was confirmed by M. Devergie (Ann. d'Hyg. 1840, p. 154), and rules were given by these chemists to distinguish *normal arsenic* from that which had been taken as a poison! The experiments of many English chemists, as well as those performed before the Academy of France by M. Orfila himself, have shown that arsenic does *not* naturally exist in any part of the animal body, and that there must have been a fallacy in his early experiments. The following is an extract from the report published by Orfila: ("*Expériences pour rechercher l'arsenic dans le corps de l'homme à l'état normal.*) Dans douze expériences faites par les commissaires avec la chair musculaire, avec des os plus ou moins calcinés et traités tantôt par l'acide chlorhydrique à l'air libre ou en vaisseaux clos, et avec du bouillon de bœuf, on n'a jamais obtenu d'arsenic (arsenic normal)." (Rapport de l'Académie Royale de Médecine, &c., par M. Orfila, 1841, p. 45.) M. Flandin showed that the effect mistaken by Orfila and Devergie for that of arsenic, probably arose from the presence of phosphite and sulphite of ammonia mixed with animal matter. (Op. cit. i. 617.) (For a full statement of the facts, see Flandin, i. 728.) If arsenic were a normal constituent of bone, the process of incineration would probably remove it. M. Legrip states that he has found what he thought was arsenic by dissolving bone in diluted muriatic acid (Journal de Chimie, Mai, 1847, p. 261); but assuming that it was arsenic, it was most probably contained in one of the acids used. It was only *after* incineration that Orfila professed to have discovered arsenic in bone. In 1840, a man was condemned to death in France on a charge of murder by poison *ten years* after the perpetration of the alleged crime. The chemical evidence against him was derived from an analysis of some of the *bones* of the deceased, in which arsenic was reported to be present by certain provincial practitioners, as well as by Orfila! (Journal de Chimie Médicale, Février, 1847, p. 82.) If it were proved that arsenic existed naturally in bones, the discovery of it in this instance could not be said to have furnished evidence of the *corpus delicti;* for by what intelligible rules could normal arsenic be distinguished from the minute portion taken into the system by absorption? Arsenic, when taken as a poison, sometimes enters into, and is deposited in bones, and it may be there detected (see ante, p. 55).

It is singular how long an error in medical jurisprudence, when once diffused on high authority, will continue to find circulation, although the experiments upon which it was based may have been long since refuted. The refutation of Orfila's opinion that arsenic existed as a natural constituent of *bone*, took place in 1841; but few cases of poisoning by arsenic even now come to trial in which the most ingenious objections, founded upon his first and incorrect experiments, are not urged to the chemical evidence of the presence of this poison. Rightly or wrongly, applicable or inapplicable, they are invariably raised by a counsel in defence. (See case of Reg. v. Richardson, Med. Gaz. xxxvii. 919.) In one instance in which I was called upon to give evidence, where about five grains of solid arsenic were found lying on the mucous membrane of the stomach, Orfila's view, that arsenic was a natural constituent of the tissues, was opposed to the chemical evidence. The inapplicability of the objection in this case was immediately made evident by the judge asking the question, whether it was possible for a human being to generate spontaneously in the cavity and upon the surface of his stomach, five grains of solid arsenic! In the case of Gilmour (Edinburgh, Jan. 1844), Dr. Christison very properly said, in answer to an objection taken to his evidence on the detection of arsenic in the liver, "that it was no constituent part of the human body, and was not formed in it. The individual (Orfila) who first promulgated this theory, only argues now that small quantities are found in the bones; but in three several experiments before the Academy in Paris, he was unable to show it." Even if arsenic existed naturally in bone, bones are either not analyzed, or they are not made the sole bases of chemical evidence. (See Lehmann's Physiological Chemistry, vol. i. p. 449.)

Arsenic in articles of food.—The detection of the poison in small quantity in the body may be assigned to the accidental presence of arsenic in food or medicine which the deceased may have taken. The *muriatic acid* which is used in making unfermented bread occasionally contains arsenic. *Vinegar* prepared by the distillation of acetate of soda with arsenical sulphuric acid sometimes contains it. The proportion of metallic arsenic found in vinegar was the 1-2200th part, or less than seven grains to thirty-five ounces. (Journal de Chimie, 1846, p. 330.) It was, however, of such a strength that it would require dilution with five or six parts of water before it would be employed for culinary or alimentary purposes: hence the quantity of arsenic present was insufficient to produce injurious consequences, or to interfere with the inferences from a chemical analysis, expect when imponderable traces are relied upon.

It has been suggested that *alum*, used in the making of bread, may sometimes contain arsenic as an impurity, and the poison thus conveyed into the body in an ordinary article of food. The alum of this country does not usually contain arsenic; and when present, as arsenic does not accumulate, it would require an enormous quantity of bread, recently eaten, to leave a perceptible trace in the body. A more important suggestion is, that *flour* is sometimes put into sacks which have had arsenic in them for the purpose of steeping corn, or corn steeped in arsenic may have been accidentally mixed with unsteeped corn. The circumstances, however, surrounding the case, generally furnish an explanation of the facts.

Seed-corn is often soaked in a solution of arsenious acid in order to destroy the spores of the fungi producing smut: it might be supposed that some of the poison would exist in the crop, and when eaten as *bread* might slowly impregnate the system. This question has not escaped the notice of chemists. M. Audouard states that he detected arsenic in the crop of corn when the seeds had been previously soaked in a solution of arsenious acid. The poison was, however, in very minute quantity. On the other hand, M. Girardin, by a satisfactory series of experiments, has proved that there is no detectable quantity of arsenic in corn under the circumstances. In some of his experiments he used more than four pounds of corn, and he could not discover in this large quantity the least trace of arsenic. (Annuaire de Chimie, 1846, 686.) This question must, therefore, be considered as settled in the negative. Independently of these facts, it must be remembered that arsenic is not an accumulative poison, that when taken in minute quantities, whatever the source, it is eliminated as fast as it is received (p. 331, ante).

The *smalt*, which is used to give a blue color to white starch, contains arsenic, derived from the cobalt ores with which it is prepared. In the case of Wooler (Durham Winter Assizes, 1855), a small quantity of arsenic was found in the wadding covering the piston of an enema apparatus. The deceased had had injections of starch during her illness; and in the desire to account innocently for the presence of this arsenic, the late Mr. Serjeant Wilkins suggested to me, subsequently to the trial, that it might have been derived from this source. I procured some of the starch supplied for the use of the deceased, and found that it was white, without any of the usual coloring, and that it contained no arsenic.

A singular question arose, on the trial of Laffarge, in reference to the supposed transference of arsenic to the living body. The quantity of absorbed arsenic extracted from the body of the deceased was estimated not to have exceeded the *one hundred and thirtieth part of a grain!* (·0077 gr.) The deceased Laffarge was a smelter of iron, and as iron frequently contains traces of arsenic, it was ingeniously suggested in the defence that this small portion of arsenic might have been absorbed into his body in a state of vapor during his attendance at the forges, and would thus account for the minute portion of poison detected by Orfila. It turned out, however, that deceased had not been near the forges for a month before the fatal symptoms appeared: therefore, as the effects were not likely to remain dormant, the poison could not be referred to this source. On the assumption that, according to the researches of M. Walchner, the oxides of iron always contain arsenic, it has been supposed the poison might find its way into the body by the employment of the hydrated oxide of this metal as an antidote in cases of arsenical poisoning. An attempt was also made to account for the arsenic found in the body of Laffarge on this ground. I have examined several specimens of the artificial oxides of iron by the processes of Marsh and Reinsch, without detecting any trace of arsenic. This exception to chemical evidence appears to me to be inadmissible. In Laffarge's case, a much better objection would have been, that the employment of large quantities of nitric acid and nitre in an iron vessel, in stewing down the whole body of the deceased, might have accounted for the minute fractional quantity of arsenic detected!

Objections of this nature can have importance only when, in the absence of symptoms and appearances, an absolute reliance is placed upon what may be termed "infinitesimal" testing. Small quantities of arsenic derived from any of these sources either occasion symptoms of poisoning, or they are speedily eliminated from the body.

Arsenic discovered in the dead body after long periods.—When the poison is really present in the stomach at the time of death, it does not easily disappear, and it may therefore be discovered for a long period after interment (ante, p. 181). White arsenic becomes slowly changed to yellow sulphuret by the evolution of sulphuretted hydrogen in the putrefaction of the stomach or its contents. It frequently forms a deep yellow stain through the coats, and appears on the external surface. I have thus found it converted to sulphuret twenty-eight days after interment (Reg. v. Jennings, Berks Lent Assizes, 1845): but the change may take place in a much shorter period. In recent cases the conversion is, however, in general only partial, as white grains may be often seen in the yellow mass. In the cases of the Cheshams (Essex Lent Ass. 1847), which I was required to examine, the coats of the stomachs were in both instances deeply dyed with large patches of yellow sulphuret nineteen months after interment. This change of color from white to yellow is not always met with, even in bodies which have been buried for a year, or longer. (Guy's Hosp. Reports, Oct. 1850, p. 206.) Care must be taken not to confound stains produced by bile on the stomach or intestines with those caused by sulphuret of arsenic.

Arsenic has been detected in a body at the end of three years (Galtier, Toxicologie, i. 370), and even after seven years (Devergie, i. 313). Wöhler is stated to have detected it in the bodies of two men seven years and six months after burial; this, however, was absorbed arsenic, and the process pursued was incineration of the soft organs by nitre. (Ann. der Chem. und Pharm. liii. 141; Chemical Gazette, 1845, 192; Med. Gaz. xxxv. 655.) In another case it is reported to have been detected after ten years. The body had become reduced to a skeleton. A confession was made by some of the parties concerned in the murder: corroborative evidence was sought for, and as it was asserted that a large dose of arsenic had been given, and the person had died in twenty-four hours, the remains of the skeleton, identified as that of the deceased, were submitted to chemical examination, when arsenic was readily detected. The examination of another skeleton, found near that of the deceased, led to a negative result: no arsenic was found. The jury were satisfied with this evidence, and returned a verdict of guilty. (Journal de Chimie Méd., Février, 1847, 82.) The particulars of the analysis are not given, nor is it stated whether the earth around the skeleton did or did not contain arsenic. Dr. Glover discovered the poison in a body after twelve years (Lancet, July 9, 1853, p. 41); and Dr. Webster, U. S., in the remains of a body after *fourteen* years' burial in a tomb (Med. Gaz. 1849, vol. xliii. p. 894). When the contents of the stomach are not allowed to drain away, the poison may be detected after a very considerable period. A person died from the effects of arsenic on the 21st of February, 1834,—the poison was at the time easily found in the contents of the stomach: these were kept for upwards of thirteen years loosely covered, and arsenic was then as readily detected in them as in the first instance,—whether Marsh's or Reinsch's process, or sulphuretted hydrogen, was employed. Arsenic, therefore, may be considered an indestructible poison.

When arsenic is discovered in the remains of persons who have died many months or years previously, it may be a question how far a medical witness is entitled to infer that death had taken place from poison. As an abstract proposition, the discovery of arsenic, even in large quantity, in such a case, would not, *per se*, prove that this was the cause of death. It would, however, furnish strong presumptive evidence, especially if the poison (absorbed arsenic) were found in the tissues of the body. This question was put at the trial of the Cheshams (Essex Lent Assizes, 1847) just mentioned. In the stomach of one child there were found about ten grains, and in the stomach of the other about twenty grains of the yellow sulphuret of arsenic. The mucous membrane beneath was of a dark red color, and the coats, which contained in every part an abundance of absorbed arsenic, were, on the whole, well preserved. I had no opportunity of examining the rest of the body, and, had the examination been made, no physical cause of sudden death operating eighteen months before would have been detected. Evidence was given to show that one child had died in about twenty-four hours after the commencement of its illness, and that it had labored under continued vomiting and intense thirst. Under these circumstances I did not hesitate to state that, in my opinion, the deceased child had undoubtedly died from the effects of poison; and no attempt was made to invalidate this opinion on the part of the defence. The learned judge who tried the case (the late Lord Denman) subsequently suggested to me that such an inference, from the mere discovery of poison, could not be drawn in all cases. Some corroborative evidence would undoubtedly be required when the poison was slow in its operation; and this would be especially necessary when the soft parts were entirely decomposed, and the arsenic was only found mixed with disintegrated portions of the skeleton. When the quantity of arsenic found in a decomposed body exceeds two or three grains, a medical witness would be justified in saying that there was a sufficient quantity of poison to account for death.

In relying upon the presence of absorbed arsenic in the tissues after long periods of interment, due allowance must be made for the effects of cadaveric imbibition. A man may die with a quantity of arsenic in his stomach only. He may have died so rapidly, that there was not time for the poison to be deposited in the organs as a result of absorption, and yet the analyst may find arsenic in the liver, spleen, and pancreas, as a result of transudation through the coats of the stomach, and refer its presence to absorption during life. This question has been elsewhere fully considered (ante, p. 69). The theory of cadaveric *imbibition* is commonly inconsistent with the circumstances under which arsenic is found. As the poison is assumed to be present in the dead body, the imbibition theory fails to explain this important fact, except by the improbable supposition that it was injected into the stomach after death. Dr. Kidd, who has advanced this theory, has himself furnished a complete answer to it as an objection to medical evidence. In his experiments, the quantity of arsenic imbibed from the stomach by the surrounding soft parts was so great, as to leave no doubt that it had been thus imparted to the organs. Absorbed arsenic is found only in very small quantity, and it is equally found in parts remote, as well as in the parts of an organ which are in proximity to the stomach. When the quantity remaining in the stomach at the time of death is very small, the amount of imbibition cannot affect the evidence; when very large, the adjacent organs are so strongly impregnated, that the analyst will know that it cannot have proceeded merely from deposition during life. It is necessary here to suggest a caution. When the organs taken from the body of a person who has died with a quantity of arsenic in his stomach are placed together in the same vessel, it is obvious that any inference regarding the presence of absorbed arsenic in the liver, or other parts, will be open to objection. In such cases there may be an impregnation of all the soft parts with arsenic in a degree proportioned to the time of contact. In the inspection of a body, the stomach and intestines should always be placed in a separate vessel, or an important branch of the medical evidence may fail.

Cemetery arsenic.—In reference to the detection of arsenic in bodies that have been long buried, another question here presents itself. Has the arsenic been carried into the body from the earth surrounding the coffin? It appears, from the researches of several toxicologists, that the soil of certain grave-yards sometimes contains a compound of arsenic, generally in an insoluble form. In eight experiments on four different soils, Orfila found three of them arsenical. He used about six pounds of earth in the analysis. As there was no sign of arsenic, except when an acid was used, he inferred that it existed in the state of arsenite, or arseniate of lime or iron. The researches of Flandin have corroborated this result; and, in one instance, this experimentalist estimated that the quantity of arsenic, in an insoluble form, in about a pound of earth, did not exceed the *twentieth part of a grain!* Admitting the existence of arsenic as a natural constituent of certain soils, of a calcareous (Gaz. Méd. Dec. 4, 1847) or of an ochreous nature (ante p. 194), it becomes important to determine how far it may affect the chemical evidence of the presence of this poison in the remains of bodies which have undergone exhumation. If the coffin be cracked or entirely destroyed, so that the earth has become intermixed with the remains, and that which surrounds the coffin yields traces of arsenic, it is evident that no reliance could be placed upon the inference that the arsenic existed in the dead body, unless the poison found in the remains was in large proportion. The reader will find cases in which doubts based upon the origin of the arsenic found in the decomposed dead body led to the abandonment of chemical evidence. (Flandin, Traité des Poisons, i. 674, 683.) When ordinary precautions are taken, a difficulty of this kind cannot, however, present itself in practice. A body buried in a coffin is rarely so far decomposed as to become covered by the soil from the disintegration of the coffin in a period shorter than from seven to ten years; and until such a complete disintegration has taken place, it is not easy to perceive how the presence of an *insoluble* arsenical compound, as a natural constituent of the soil, can present any objection to the results of an analysis. In the examination of these soils, it has been clearly ascertained that no arsenical compound, *soluble in water*, has existed in them; therefore, if distilled water should yield, on boiling the remains, a solution of arsenic, and the earth from the grave does not, the arsenic could not have been derived from the soil. It has been supposed that the arsenic may have been carried by percolation from the soil into the body; but in this case, as Flandin has observed, the exterior of the body would contain more than the interior; while in a case of arsenical poisoning (except when dependent on local application) the liver and stomach would yield more than the skin. (See Galtier, i. 368; Flandin, i. 429, 741.) M. Devergie thinks that a body buried without a coffin, and covered simply by a shroud, might thus, under the access of water, imbibe arsenic from the soil: and such is the opinion of M. Van den Broeck, even when the compound of arsenic is perfectly insoluble in boiling water. (Flandin, i, 442.) This opinion is, however, directly opposed to well-observed facts.

This question at one time acquired some importance from the result of the trial of Elizabeth Johnson for poisoning her husband with arsenic. (Liverpool Lent Assizes, 1847.) The accused, concerning whose guilt, morally speaking, there could be but little doubt, appears to have owed her acquittal entirely to the assumption that arsenic, in a *soluble* form, might have found its way into a dead body through a crack in a coffin, although it had not been shown that the soil of the churchyard where the body was buried contained any trace of the poison, either soluble or insoluble! Still, so far was this scientific question carried, that the assumption of arsenic being present in the soil was allowed in favor of the prisoner. The deceased died on the 3d of December, 1846. The evidence that he had died from arsenic was not then rendered complete by the examination and analysis. The body was exhumed, after *three months* interment, on the 9th of March, 1847; and Mr. Watson, of Bolton, examined the viscera. This gentleman clearly detected arsenic in the liver, intestines, heart and blood, kidneys, gullet, and tongue, and in the muscular substance of the right thigh. The largest proportion of poison was derived from the liver, kidney, and intestines; and the *smallest quantity*, amounting to only an exceedingly slight trace, was detected in the heart and *blood*.

Mr. Leigh, a medical witness, who was present at the exhumation, stated that there was a large quantity of "*fluid blood*" in the body. When asked by the learned judge (now deceased) who tried the case, to account for this, he said he thought it might have arisen from the penetration of water, as the grave was wet, and the coffin was split from one end to the other. The body had only been buried three months; but the wood was very thin, and had apparently cracked from the superincumbent weight of earth. The judge then asked, "Supposing there was arsenic in the soil of the churchyard, was it not possible for some of that arsenic to be washed into or introduced into the body along with the water?"—to which Mr. Leigh replied, he thought it was possible. This statement, so materially affecting the chemical evidence, was allowed to pass to the jury as a probable mode of accounting for the presence of arsenic, not in the blood only, but in the liver, kidneys, intestines, and even *the tongue, œsophagus, and muscles of the thigh!* The first intimation which Mr. Watson received of the mode in which his chemical evidence would be applied, was in the charge of the learned judge to the jury: and there can be no doubt that from arsenic not having been clearly detected in December, the jury referred its discovery in the dead body in March to the series of assumptions above detailed. As the *smallest* quantity of arsenic was found in the *blood*, which it was assumed was mixed with the cemetery arsenical water (the supposed actual solvent of the poison), it was utterly impossible to refer the presence of arsenic in the other viscera of the body to impregnation from this water. To complete the history of the case, Mr. Watson, to whom no questions on the subject were put at the trial, although he was the witness who really discovered the poison, informed me that he subsequently procured a quantity of earth from the churchyard in which the body had been buried, and there was not in it a trace of arsenic, either in a soluble or insoluble form! That arsenic is not thus washed into a dead body buried in an arsenical soil, is not a mere speculative opinion—it is based on fact. In 1844, M. Ollivier made the following communication to the French Academy. The body of a female, alleged to have been poisoned by her husband, was exhumed: it was ascertained that no poison was present, and that she had died from natural causes. A married woman, who wished to marry this man, poisoned her husband, and arsenic was detected in his body (the liver), as well as in the earth of the cemetery in which it was buried. From a suspicion that the analysis had not been carefully made, the body of the female, which had been buried in the same cemetery, and which, it appears, on re-interment, had accidentally fallen out of the coffin into the grave, and had become completely covered with the arsenical earth, was again exhumed after several months had elapsed. Not a trace of arsenic, however, was detected in it. (Lancet, Aug. 17, 1844, p. 638.) Orfila having procured a large quantity of earth taken from the cemetery of Epinal, which was known to be impregnated with arsenic, buried in it a full-grown fœtus, the liver of an adult, and various portions of dead human bodies. *Three months* afterwards these various parts were exhumed, and were found to be in a complete state of putrefaction. They were carefully examined for arsenic by the usual processes, but not a trace of the poison could be detected. (Acad. of Med. 29th June, 1847; Gaz. Méd. 3d July, 1847, p.

535; also Ann. d'Hyg. 1847, ii. p. 194.) Hence it appears evident that, under the most favorable circumstances, the dead human body does not acquire an impregnation of arsenic from contact with arsenical earth. The mode in which medical evidence was dealt with at the trial of Johnson was not only in violation of all probability, but directly opposed to ascertained facts! Another case, in which a question arose respecting the presence of arsenic as a constituent of the earth of cemeteries was that of the Queen v. Richardson. (Med. Gaz. xxxvii. 919.) Its presence in the earth was here clearly negatived by the medical witness, who had taken care to make an analysis of it before the trial.

In a series of poisonings in Norfolk, the investigation of which was conducted by Mr. Firth, in the summer of 1846, it was proved that the soil of Happisburgh churchyard, in which six of the bodies were buried, contained arsenic, although taken at a distance of three feet from the graves of the poisoned family! Mr. Firth informed me that the poison was easily detected in half an ounce of the earth by the process of Reinsch. He could not procure any trace of it by acting on the soil with boiling water, but readily by using muriatic acid as a solvent. This proved that the arsenic was, as represented by the French chemists, in an insoluble form. One of the bodies had been buried eleven years; therefore, this discovery of arsenic in the soil was really of practical importance. A mass of the decomposed remains was scraped up with a spoon from the sides of the lumbar vertebræ; and arsenic was found by Reinsch's process, but in very unequal quantity in equal parts of the remains. Mr. Firth, knowing that the soil of the churchyard contained arsenic, referred the poison to this source, and not to any introduced into the body during life. This was a very proper precaution, in reference to remains so completely decomposed, and in contact with the soil, notwithstanding a suspicion of death from poison in the case of the deceased. The observations of Mr. Firth on the insoluble state of the arsenic in the earth around the decomposed remains of the deceased, appear to furnish an answer to an ingenious speculation which has been advanced, namely, that ammonia, generated by putrefaction, might act as a solvent to the arsenical compound, and, under the percolation of water, convey the poison into the dead body. (Gaz. Méd. 12 Juin, 1847, 452.) It is only in the latter stages of decomposition that a body is likely to be so exposed to the soil as to render it possible for the cemetery arsenic to be transferred to it; but then the production of ammonia will have ceased, and the soft parts will have become destroyed. The presence of hydrosulphuret of ammonia appears to exert but little influence, since the speedy conversion of the poison to sulphuret tends to fix it permanently in yellow patches in the substance of the organs. I have thus discovered it in large quantity nearly two years after interment, in a stomach abounding with hydrosulphuret of ammonia.

There are many speculations regarding the source of the arsenic in the soil of cemeteries. From the researches of M. Walchner, it appears that arsenic is a constituent of all soils abounding in oxide of iron, and in all the ochreous deposits of acidulous waters. It is also naturally contained in some mineral waters, under the form of arseniate of lime. He has discovered it in all kinds of clay, marl, or earthy deposits colored by oxide of iron, and he believes it to be universally diffused. (Comptes Rendus, Sept. 21, 1846, p. 612; also Brit. and For. Med. Review, July, 1855, p. 232.) On this view, it may exist in other localities as well as in the soil of churchyards; and would only be found in the latter when the earth was of a highly ochreous character. Some kinds of pyrites contain arsenic, with sulphur and iron. It is not improbable that, by the slow decomposition of this substance, oxide of iron, sulphate of iron, and arseniate of iron, may result. This may become diffused through the soil and render it arsenical. I have examined the earth of cemeteries on four different occasions, in which arsenic was found in exhumed bodies. In no instance was there arsenic in the earth of the grave.

When food or vomited matters containing arsenic have been thrown on the earth, this question may incidentally present itself as an objection to medical evidence. In a case which was the subject of a trial in France (*Affaire Malaret*, May, 1846), it appeared that the wife, who was charged with the murder of her husband, had thrown some of the vomited matters out of a window. The ground beneath was examined, and arsenic was found in the earth by the wall immediately under the window, and in smaller quantity for a circuit of more than two yards. Beyond this space the earth did *not* contain arsenic. The poison was found in the tissues of the body, which had been for some time buried, and was exhumed for the analysis. The earth of the graveyard was examined, and contained no arsenic. (Gaz. Méd. 20 Juin, 1846, p. 498; and Ann. d'Hyg. 1847, i. p. 400.) A similar question arose in the case of Reg. v. Lucas and Reeder (Cambridge Lent Assizes, 1850). The deceased had died from some effects of arsenic. One of the sources of proof was derived from the analysis of some earth taken from a garden-path, on a spot where the deceased had been seen to vomit in the early part of her illness. I found arsenic in a few ounces of this earth, in a soluble form, and in rather large proportion. In earth taken from the path at a distance from this spot, no arsenic was found, either in a soluble or insoluble form.

In analyzing the earth of a cemetery for arsenic, a decoction of one or two pounds in water should be made. This may be poured off from the undissolved residue, filtered and examined by the processes of Reinsch or Marsh. If this fails to reveal arsenic, the residue may be digested in hydrochloric acid diluted; the solution filtered, the iron and lime precipitated by ammonia and the oxalate of ammonia, and the residue, after evaporation, may be tested for arsenic by either of the processes above mentioned. The precipitated oxide of iron must also be tested, as the arsenic, if present, may have been partly or wholly precipitated in combination with the hydrated oxide.

Preservative effects of arsenic.—It has been observed by those who have had opportunities of inspecting exhumed bodies in which arsenic has been found, that the viscera were frequently in a remarkable state of preservation. I have noticed this in cases in which bodies have been interred for periods varying from a fortnight to two years. This singular effect has been ascribed to an antiseptic or preservative property in arsenic. In poisoned bodies, the viscera, which are the seat of poison, retain their firmness and characteristic appearance in a striking degree, so that even the effects of arsenic during life,—inflammation and ulceration,—may be observed (ante, p. 157). In the case of Mrs. Bacon (Lincoln Aut. Assizes, 1857), whose body was disinterred after nearly two years' burial,—the head, chest, arms, and legs, were soft, of a dark brown color, and much decomposed. The abdomen and its coverings were so preserved, that the muscles retained the redness and firmness seen in a body a fortnight after death. The coats of the stomach and intestines were also firm, and presented marks of inflammatory redness. Arsenic in small quantity was found in these parts. Several similar instances of the preservation of bodies, exhumed at shorter periods, have presented themselves to me, and this is in accordance with the experience of others. The parts preserved are commonly those in which the arsenic is either contained, or has been contained. It is not correct to say that the bodies are not putrefied: ammonia, and sometimes hydrosulphuret of ammonia, has issued in large quantity from stomachs, in the contents of which arsenic was found; but the course and rapidity of ordinary putrefaction are modified or changed. To this state, Dr. Geoghegan has given the name of rancid putrefaction. (Dublin Quarterly Journal, Feb. 7, 1851, p. 110.) He has found that a preservative influence is visible in the viscera of those who have been poisoned by arsenic, even after the whole of the arsenic has been eliminated. I have noticed the fact, that in a poisoned body the degree of preservation of parts has been by no means in proportion to the quantity of arsenic present. It has been as great where the arsenic has been found only in traces, as where it existed in the form of a crystalline powder. Dr. Geoghegan assigns this to a catalytic power in the arsenic: and, beyond doubt, there are cases in which the properties of a body are chemically changed by the influence of a substance, and when the change is once effected, the substance may be withdrawn, but the original properties are not restored.

This preservative influence extends also to the food in the stomach at the time of death. I have thus been able to recognize, by the aid of chemistry and the microscope, the nature of the last meal which the deceased had taken after the body had been lying many months in the grave. This evidence is sometimes of importance in corroborating or contradicting the statements of witnesses. We must be careful, however, not to strain it, and infer, because arsenic is mixed with the food, that this was the actual vehicle in which it was administered or taken.

In some undoubted cases of poisoning by arsenic, putrefaction has advanced, apparently without having been checked by the presence of the poison; and even when, in the first instance, putrefaction was retarded, Dr. Geoghegan has noticed that, in the last stage, the arsenic has itself partaken of the change, and has escaped under the form of arsenuretted hydrogen. (Dublin Journal, Feb. 1851, p. 110.) A medical witness, therefore, must not place undue reliance on the condition of an exhumed body. There are instances in which, without reference to the influence of arsenic or any intelligible cause, a dead body is found, some months after burial, in an unusual state of preservation. The retardation of putrefaction, when arsenic is found in an exhumed body, may be recognized as a corroborative indication of poisoning; but the medical inference cannot, it appears to me, be carried further.

CHAPTER XXV.

ARSENICAL VAPORS—ARSENITES OF POTASH AND SODA—SHEEP-WASH—ARSENITE OF COPPER—POISONING BY GREEN CONFECTIONERY—ALLEGED EFFECTS OF ARSENICAL PAPERS—FLY-POWDER—ARSENIC ACID—ARSENIATES OF POTASH AND SODA—ORPIMENT—SULPHURETS OF ARSENIC—CHLORIDE OF ARSENIC—ARSENURETTED HYDROGEN.

ARSENICAL VAPORS.

THE vapors which escape from the arsenic and copper-smelting works of Cornwall and South Wales, are those of arsenious acid, or white arsenic. From the flues the arsenic issues as a thick, white vapor; and, when no precautions have been taken, it has destroyed cattle, as well as vegetation, to a great extent. In the grinding mills it may be respired as a fine dust. Great precautions are taken by the workmen, by plugging the nostrils and covering the mouth, to avoid respiring this arsenical dust; but, in spite of these, accidents are liable to occur. The men who remove the impure arsenic from the flues suffer from severe pustular and scaly eruptions, affecting the scrotum and other parts of the skin where there are hollows or depressions. Thus, according to Dr. Jago, the parts especially liable to be attacked next to the scrotum, are the depressions between the lower lip and chin, the angles of the nose and face, and the lines along the forehead; in fact, every crevice in which the arsenical dust can lodge. This appears to indicate a local irritant action. Among the constitutional effects there have been noticed—cough, with shortness and difficulty of breathing, debility, emaciation, profuse perspiration on slight exertion, scantiness of urine, and severe pulsations of the heart. The tongue presents red edges, with a white fur; and the gums are inflamed. Frequent nausea and vomiting are also among the symptoms. (See paper by Mr. Kesteven, Assoc. Med. Jour. Sept. 1856, p. 811.) A few years since, an inquest was held on the body of a child at Plymonth, whose death was referred to arsenical fumes. A case has been elsewhere related (p. 216, ante) in which death was erroneously referred to arsenical fumes escaping from burning minerals. In Cornwall, the deaths of workmen may sometimes be traced to the poisonous vapor; but, on the whole, considering the nature of their occupation, the men enjoy average health. Many cannot work long in the arsenic factories, while others have continued to work in them for twenty or thirty years.

In the case of the Queen *v.* Garland (Cornwall, Lent Assizes, 1851)—a prosecution for nuisance and damage from arsenic works,—it was proved that animal and vegetable life suffered, to a great extent, from these fumes. Horses and cattle perished, and, before death, they became much emaciated, and lost their hair. It would appear that these effects are produced as much by the animals pasturing on poisoned herbage, as by the actual breathing of the arsenical vapors. After death, the stomachs and bowels were found inflamed, and sometimes mortified. Donkeys were especially liable to suffer, owing to these animals eating thistles and plants with hard and irregular surfaces, favorable for the retention of the arsenical dust. In South Wales, the animals within the range of the arsenical smoke from the copper works have, in addition to other symptoms, suffered from enlargement and diseases of the joints. These facts, it will be perceived, are quite adverse to the Styrian hypothesis of the fattening effects of arsenic on horses and cattle (ante, p. 93).

ARSENITE OF POTASH. (FOWLER'S SOLUTION.)

All the compounds formed by arsenious acid with the alkalies are poisonous. Those of potash, soda, and ammonia, are soluble in water, and therefore act with more energy. The ARSENITE OF POTASH is used in medicine, and is well known under the name of FOWLER'S MINERAL SOLUTION, or Tasteless Ague Drop. It is made by boiling arsenious acid with carbonate of potash, the latter being in slight excess, and it is colored with compound tincture of lavender. In the preparation of the London Pharmacopœia, there are four grains of arsenious acid in a fluidounce (or eight fluid-drachms) of the solution (1-120th gr. in one minim). Its real strength may be affected by any impurities in the arsenious acid employed. The preparation used in Scotland is of the same strength; but that of the Dublin College is rather stronger. The action of this liquid as a poison, in large doses, is in all respect analogous to that of arsenious acid. It acts more energetically by reason of the poison being in a perfect state of solution.

Symptoms and Appearances.—There is, so far as I know, only one case recorded in which this solution has destroyed life. (Provincial Journal, June 28, 1848, page 347.) A woman took half an ounce of the solution (= two grains of arsenic) in divided doses, during a period of five days. There was no vomiting or purging; but after death, the stomach and intestines were found inflamed. The solution is much used by the poor in the Fen districts of Cambridgeshire, as a preventive of ague. It has occasioned symptoms of poisoning when given in an overdose, but I have not heard of any case proving fatal. This domestic use of arsenic in these districts, may, however, account for the occasional detection of arsenic in a dead body, irrespective of poisoning.

That Fowler's Solution is a powerful agent, and that the boundary between its medicinal and poisonous action is very slight, will be sufficiently indicated by the cases elsewhere reported (ante, page 340).

There is one form of poisoning by an alkaline arsenite which it is desirable to point out. A mixture of arsenic, soft soap, and tar-water, is largely used in agricultural districts for killing the fly in sheep. In June, 1858, a woman swallowed a quantity of this liquid. She soon afterwards experienced severe pain in the stomach, nausea, and violent vomiting, followed by purging, great heat in the throat and stomach, with intense thirst. She died in twenty-four hours after taking the poison. The mucous membrane of the stomach presented a few patches of inflammation, but was of a pale bluish color in other parts. The lining membrane of the gullet, at the part where it entered the stomach, was of a bright pink color. The duodenum and upper part of the small intestines were highly inflamed. A portion of the liquid which deceased had taken was found to be highly alkaline, and smelt strongly of tar. It was a saturated solution of arsenite of potash, with excess of carbonate. When paper, impregnated with the liquid, was burnt, a white smoke was evolved, which, when received on a cool surface of glass, gave the usual indication of arsenious acid with ammonio-nitrate of silver. The symptoms and appearances were similar to those observed in poisoning by arsenious acid.

Dr. Mitchell lately met with a case in which a mixture of arsenic and soft soap, applied locally, produced all the well-marked effects of poisoning by arsenic, as well as an intense local action. A man applied this mixture to his scrotum and armpits for the purpose of killing pediculi. In twelve hours he began to feel a stiffness in the neck, and a slight difficulty in swallowing. The cuticle of the scrotum peeled off, leaving the cutis inflamed and bleeding. There was great thirst, with headache, and a sensation as if the hair was being pulled up by the roots. There was irritability of the stomach, with vomiting, purging, and pain on pressure. He said that he felt as if his bowels were on fire. Under treatment this man recovered. (Med. Times and Gazette, Dec. 10, 1853.) Shepherds who have used this solution for dipping sheep, have occasionally suffered severely from symptoms of irritant poisoning as well as from eruptions on the skin.

Orfila refers to a singular case of poisoning by a compound arsenite of potash and lime in a solid form (*i. e.* as a soap) in which the most marked nervous symptoms (trismus) appeared in three quarters of an hour: the individual recovered. (Toxicologie, i, 449.)

Fatal Dose.—The medicinal dose of Fowler's Solution is from four to ten minims twice a day. It is common to commence with four to five minims, and gradually increase the dose. Dr. Pereira has known fifteen minims to have been taken three times a day for a week without ill effects; and Dr. Mitchell, of Ohio, has given from fifteen to twenty drops three times a day in intermittents. (Materia Medica, i, 718.) In some persons there is a strong idiosyncrasy with respect to arsenic (see ante, p. 339); and even smaller doses than those commonly prescribed can hardly be borne without causing alarming symptoms. A case was reported, in the Pharmaceutical Journal for 1845, in which one drachm (equal to half a grain of arsenic) was taken with comparative impunity.

According to Mr. Bullock, the Pharmacopœial preparation is not a true arsenite of potash, but a solution of arsenious acid in carbonate of potash, with a minute trace of the arsenite. (Lancet, Dec. 21, p. 674.) The uncertainty of its composition may possibly account for the variable effects produced by this liquid. Mr. Hunt, who has largely employed arsenic in the treatment of skin diseases, states that when the susceptibility is not great, a dose of *two drachms* of the solution (= *one grain* of arsenic) can be borne about as well in *one* dose as in twenty. He quotes a case in which a patient took *two drachms* of this solution (= one grain of arsenic), in twenty-four hours, by mistake. It cured the ague, for which it was prescribed, and had no injurious effect. (Med. Times, September 14, 1850, page 270.) It is difficult to explain such anomalies by varying susceptibility only; they are more probably due to the uncertainty of composition in the preparation employed.

The *treatment* of a case of poisoning by a soluble alkaline arsenite would be the same as that for arsenious acid; but the hydrated sesquioxide of iron might be given with a greater prospect of benefit.

Analysis.—This solution has the odour of tincture of lavender, is of a reddish color, and has an alkaline reaction. It gives at once a green precipitate (arsenite of copper) with the sulphate of copper, and a yellow precipitate with nitrate of silver. Acidulated with muriatic acid, and treated with a current of sulphuretted hydrogen gas, it yields a yellow sulphuret; and when boiled with muriatic acid and copper, a deposit is obtained which readily furnishes octahedral crystals of arsenious acid. (See REINSCH'S PROCESS, ante, p. 358.)

The only instances which I have known of poisoning by the *arsenite of soda* are the cases referred to at p. 341, ante, in which three hundred and forty children had this poison administered to them through culpable negligence, at their morning meal. The effects show that, like the arsenite of potash, it is a powerful poison. The emetic and purgative treatment was effectual in every case, without resorting to the use of hydrated oxide of iron.

ARSENITE OF COPPER, SCHEELE'S GREEN.

The poisonous properties of this compound are undoubtedly due to the arsenic which it contains; hence it may be appropriately considered with the arsenites. It is the only metallic arsenite which is met with in commerce and the arts, and it constitutes, wholly or in part, a great variety of green pigments,—known as Emerald green (aceto-arsenite of copper), mineral green, Brunswick, Schwemfurt or Vienna green, &c. It is thus found in the form of oil-paint in cakes in boxes of water-colors, spread over confectionery, in wafers, in adhesive envelopes, and lastly and most abundantly, in the various kinds of green decorative papers for covering the walls of sitting and bed-rooms. A manufacturer has informed me, that so great was the demand for this "cheerful" but poisonous color, that his average consumption of arsenic had amounted to about two tons weekly! The extensive diffusion of arsenic in this form, with the facility which it gave for universal poisoning when the knowledge and inclination coexisted, was actually converted into an argument to show the inutility of placing any legal restrictions on the sale of arsenic or other poisons! The Prussian government, after instituting inquiries, has solved the question, by prohibiting the manufacture, sale, and use of these arsenical papers. (See ante, p. 329.) In this country, however, the arsenical green decorative papers are unconsciously purchased by the public, and are most extensively used in dwelling-houses by reason of the cheapness and durability of the green colors! We may first consider the effects of Scheele's green in its ordinary state of powder.

Symptoms and Effects.—Although the arsenite of copper is insoluble in water, it is sufficiently soluble in the acid mucous fluids of the stomach to be taken up by the absorbents, and carried as a poison into the blood. A child, aged three years, swallowed a small capsule of Scheele's green, used by his father as a pigment. In half an hour he complained of violent colic: there was frequent vomiting, with purging, cold sweats, intense thirst, and retraction of the parietes of the abdomen. The mouth and throat were stained of a deep green color. Hydrated sesquioxide of iron was given: in about an hour, the vomiting and purging ceased, and soon afterwards the thirst and pain in the abdomen abated. The next morning the child was well. In another case, a child, a year old, ate several pieces of a cake of arsenite of copper used for colors. There was immediate vomiting of a liquid containing green-colored particles of the arsenite, but there were no other urgent symptoms. White of egg, with sugared water, was given to it. After a short time the child became pale, and complained of a pain in the abdomen: the pulse was frequent, the skin cold, and there was great depression. Copious purging followed, soon after which the child recovered. (Galtier, i, 636.) In the cases of two children poisoned by confectionery colored with this substance, the chief symptom was incessant vomiting of a light green-colored liquid, resembling bile diluted with water. Mr. Bulley, of Reading, who reports these cases (Medical Times, April 28, 1849, page 507), describes the symptoms as severe, although the quantity of poison swallowed was small. Under the use of an emetic of ipecacuanha the children recovered. A case was communicated to me in July, 1849, by Mr. J. B. Hicks, in which a child, aged seven years, ate a slice of cake with a part of a green ornament on it. There was severe pain, with thirst, and a burning sensation in the throat, with constant vomiting, but no purging. The child recovered in three days. The green pigment was found to be pure arsenite of copper mixed with sugar. (Guy's Hosp. Reports, Oct. 1850, p. 218; see also Medical Gazette, vol. xliii, p. 304.) In two cases which I examined in January, 1853, a small quantity of a confectionery ornament, colored with arsenite of copper, proved fatal to two children. The symptoms and appearances were those of poisoning by arsenious acid. The quantity taken could not have been above two or three grains. The children picked up the ornament in the street, and shared it between them. The poison was spread over a layer of sugar!

M. Chevallier relates a singular case, in which arsenite of copper was used by a pork-butcher, for ornamenting a boar's head supplied at a breakfast given on a festive occasion by an eminent Parisian lawyer. The head was decorated most artistically with masses of fat, which were colored red and green. One of the guests, well acquainted with chemistry, was struck with the rich green color of the fat, and reserved a portion for a private analysis. He found the coloring matter to be pure arsenite of copper, forming about two per cent. of the weight of the fat! It appeared, on inquiry, that notwithstanding the police regulations, the butcher's boy had procured the poisonous compound at a neighboring color shop. (Journal de Chimie Médicale, Janvier, 1847, p. 16.)

Dr. Geoghegan informed me that an accident occurred in Dublin in 1842, by which fourteen children suffered from symptoms of poisoning, in consequence of their having eaten some confectionery ornaments colored with arsenite of copper. In two or three of the cases jaundice followed. The dangerous practice of using this powerful poison to give a color to confectionery was at one time very prevalent, but it is not now so frequent. An instance was communicated to me in which three lives were nearly sacrificed at a school near Manchester, owing to the boys having eaten some ornamented confectionery, which owed its green color to arsenite of copper. They suffered from violent vomiting, severe pains in the stomach and bowels, and spasms in the extremities. Three animals which ate of the vomited matters were attacked by similar symptoms.

In a case which was the subject of a criminal trial, this deadly compound was proved to have caused the death of a gentleman by reason of its having been employed to give a rich green color to some blanc-mange served at a public dinner:—the person who employed it considering that emerald or mineral green was nothing more than an *extract of spinach!* It led to death under the usual symptoms, and the parties were convicted of manslaughter and sentenced to imprisonment. (Reg. v. Franklin and Randall, Northampton Summer Assizes, 1848.) Most of the colors used for confectionery are of a poisonous nature: the pink color given by cochineal or madder is the only one which can be regarded as innocent.

Dr. Traill met with a case in which a child aged three years suffered severely from symptoms of arsenical poisoning, owing to its having sucked some slips of paper colored with this green pigment; some of the paper, still retaining a green color, was passed in the motions. The child recovered (Edinburgh Monthly Journal, July, 1851, p. 1).

Among other uses of this noxious compound, we find it employed for imparting a bright green color to the shelves of bakers' and greengrocers' shops. An incident which occurred to myself will show that food may thus acquire an arsenical impregnation. Several loaves of bread were supplied to me, having upon the undercrust a quantity of green-colored pigment, which on analysis turned out to be arsenite of copper, containing about fifty per cent. of arsenic! On inquiry, I found that the baker had recently painted the shelves of his shop with this pigment, and the hot loaves placed upon them had taken off a portion of the arsenical paint. It is easy to conceive that an accident of this kind, if undetected, might lead to serious results, and perhaps to very erroneous suspicions. (Medical Times and Gazette, April, 1854, p. 326.)

Another alleged form of poisoning by this substance which has attracted some attention, is in the state of vapor or fine dust applied to the membrane of the lungs, or in the state of powder as applied to the skin. In the former edition of this work the following case was related. A young man, after having been engaged for nine days in printing with an arsenical green pigment, was seized with irritation and watery discharge from the nose, swelling of the lips and nostrils, and headache. The next day he experienced severe colic, and great muscular weakness: but these symptoms disappeared in about eight days. It is probable that, in this case, the arsenite of copper had been taken into the body in the state of fine powder. (See Arsenical Vapors, ante, p. 382.) I have since been informed, that the persons who manufacture and hang the colored paper on walls, suffer from boils, inflammation of the eyes, and other symptoms of irritation. In one case now under treatment, pustular tumors have shown themselves on the wrists and ankles, and there is excessive sensitiveness and irritability of the skin. If removed they soon recover. In a former page I have alluded to the mysterious deaths of a whole family (see cases of the Arzone family, ante, p. 119). The father was a pigment-manufacturer, and there is great reason to believe that he and his family fell victims to the respiration of arsenical dust or vapors. According to M. Bouchardat (Annuaire de Thérapeutique, 1846, p. 209), the workmen who handle the emerald-green in making the papers, are subject to serious disorders of health. They sometimes suffer from eruptions of the skin,—one of the local effects of poisoning by arsenic (see Assoc. Med. Journal, 1856, Sept. 6, p. 757; Sept. 20, p. 810, and ante, p. 335), with œdema (watery swelling) of the face, and boils frequently forming in the scrotum. There is irritation with discharge of fluid from the mucous membrane of the nose, and abundant salivation. In the more advanced stage, there are colicky pains, headache, and prostration of strength. (See Ann. d'Hyg. 1847, ii, p. 56; and Journal de Chimie, Juillet, 1858, pp. 394, 397.)

More than ten years since, Dr. Martin announced that the use of this arsenical green in oil-paint had an injurious effect upon those who inhabited apartments recently painted with this substance. Four pounds of Scheele's green had been used in painting the walls of a low damp room. In a few days a putrescent and highly disagreeable odor was perceptible. When the windows were closed, those who remained in the room experienced headache, pain in the chest, and other disagreeable symptoms. The color was scraped from the walls, and the room was then inhabited without any of these unpleasant symptoms being observed. Dr. Martin attributed the effects to the production of arsenuretted hydrogen. The poisonous salt may, however, have been itself carried off in vapor, like white lead, under similar circumstances, by the oil of turpentine. In a note attached to this case, it is stated that since the mixed acetate and arsenite have been substituted for carbonate of copper in painting the walls of rooms, many persons who have slept in rooms painted green, have complained in the morning of headache, nausea, dryness of the mouth and throat, and cough. The symptoms went off during the day. In one instance the foul odor was referred to mice, and the wainscot was about to be removed, when a suspicion arising that it was owing to the green color used as a pigment, this was removed, and the smell disappeared. (Gaz. Méd. 13 Fev. 1847, p. 130.)

I have elsewhere referred to the probable effects of wall-papers loosely covered with the aceto-arsenite of copper (ante, p. 329). This pigment contains fifty-nine per cent. of arsenic, and from some of these papers the noxious material may be easily scraped or removed by friction. A square foot of the paper may yield from twenty-eight to seventy grains of the arsenical pigment, and in rooms exposing five or six hundred square feet, a large quantity of arsenic is thus distributed over an extensive surface. Dr. Hinds, of Birmingham, noticed, that in occupying a room which was covered with a wall-paper of this kind, he suffered from severe depression, nausea, pain in the abdomen, and great prostration of strength. These symptoms appeared on every evening that he sat in this room: this led him to suspect that they were connected with the room, and on examining the paper he found in it a quantity of arsenic. (Lancet, 1857, vol. i, p. 193.) Two other cases occurred in his practice, where similar symptoms were produced in a man and his wife, under similar circumstances. To these I have elsewhere referred (ante, p. 330). Dr. Halley, of Harley Street, suffered from constant headache, dryness of the throat and tongue, with internal irritation. In about three weeks, he became completely prostrated, and was threatened with paralysis of his left side. He called on me and described his symptoms, bringing, at the same time, a portion of the wall-paper of the room in which he was in the habit of sitting; and this I found to be loaded with arsenic. He removed the paper, and since then has recovered his health. Several cases have since come before me, in which, whether real or imaginary, symptoms of a *similar* kind have been referred by persons to the habitation of rooms papered with the arsenical green. At the same time, there have been many cases in which the occupation of rooms thus prepared has been attended with no injurious effects. This fact, as well as the result of a few imperfect experiments, have led some persons to affirm that the arsenical papers have not produced the effects ascribed to them. (See Pharmaceutical Journal, April, 1858, p. 520, and May, 1858, p. 554.) The connection of cause and effect, however, appears to me to be too plainly made out in the cases of Dr. Hinds and Dr. Halley, to be set aside as a mere coincidence. The symptoms in both cases were similar, and such as arsenic is well known to produce: there was no other source of arsenic, and no other cause to explain them; and they entirely ceased on the removal of the arsenical paper. It may not be easy to detect arsenic in the air of a room thus papered, but then it is equally difficult to detect lead in the air of fresh painted rooms, in which persons have been paralysed, by passing a night! In the year 1837, and subseqently, arsenic was largely used in the manufacture of a spurious kind of wax-candle. The workmen suffered from boils and other disorders, and some who occupied rooms in which such candles were burnt, complained of symptoms like those of arsenical poisoning. As in reference to the arsenical papers, it was alleged,—because arsenic could not be detected in the air of a room, and all persons did not suffer from the use of the candles,—that the illness was owing to some other cause. Although there are difficulties in explaining how it happens that more accidents do not occur, it appears to me there is already sufficient evidence to justify an enforcement of the Prussian regulation, prohibiting the use of arsenic for such a manufacture, or in allowing the paper to be sold only on the condition that the words "arsenic, poison," are stamped upon it! (See Pharm. Journal, May, 1858, p. 553.)

Dr. Böcker, of Bonn, one of the most recent writers on Toxicology, refers to the effects of chronic poisoning produced on persons inhabiting rooms of which the walls are covered with arsenical paper-hangings, and states, that on several occasions he has been called upon to treat such cases. A removal of the cause has generally proved sufficient. Dr. Böcker considers that a damp state of the wall renders them injurious. (Die Vergiftungen, 1857, p. 132; also Casper's Vierteljahrschrift, Januar, 1858, p. 184.)

Analysis.—(SCHEELE'S GREEN.) This salt is of a green colour, the depth of which is modified by admixture with other substances. It is insoluble in water, but soluble in ammonia and in acids, forming a blue solution. When very gently heated in a reduction-tube, arsenious acid is sublimed in minute octahedral crystals. These may be collected, dissolved in water, and tested in the usual way. (See ante, p. 348, Exp. 7.) The residuary oxide of copper may then be dissolved in nitric acid and tested. With charcoal powder, the arsenite gives, although with some difficulty, a ring of metallic arsenic: but the arsenical nature of the salt is easily determined by boiling it with diluted muriatic acid and a slip of metallic copper or copper gauze. (See Reinsch's Process, ante, p. 358.) Metallic arsenic is immediately deposited on the copper. When the arsenite of copper is used in confectionery, the substance upon which it is spread is either soluble (sugar or starch) or insoluble (plaster of Paris). In either case we scrape off the green color and digest it in a small quantity of water. In the first case the arsenite of copper is deposited, while the sugar or starch is dissolved; in the second, the arsenite of copper is deposited with the sulphate of lime. The former may be separated from the latter by ammonia, and re-obtained pure by evaporation. Should the arsenite be mixed up with fat or oil, it will easily subside as a sediment on keeping the substance melted, and the deposit may be freed from any traces of fat by digesting it in ether. The pigment called EMERALD GREEN is a mixture of arsenite and acetate of copper. The presence of arsenic in this compound is easily detected by muriatic acid and metallic copper.

METALLIC ARSENIC. FLY POWDER.

It is generally considered that metallic arsenic is not poisonous; but, as this metal is easily oxidized, it speedily acquires poisonous properties. According to Berzelius, the metal is slowly converted, by exposure to the air, to a pulverulent suboxide of a black or a brownish-black colour. This is commonly called "fly-powder,"—a name also applied to the arsenical cobalt ores reduced to powder. Thus, what is called the "Tunaberg ore"—a mixture of cobalt, arsenic, iron, and sulphur—is largely used on the Continent under the name of "fly-powder;" and as it comes within the reach of children, it frequently gives rise to accidents.

Symptoms and appearances.—A few years ago, Dr. Schobbens was called to a man who had taken some of this powder by mistake for a purgative. He was soon attacked with the usual symptoms of poisoning by arsenic. He swallowed a large quantity of milk, which occasioned immediate vomiting. As fifteen hours had elapsed before a medical man saw him, no treatment was of any avail, and he died from the effects of the poison. In another case a child, aged 4 years, swallowed a portion of fly-powder. The hydrated sesquioxide of iron was given every half-hour, and the child recovered the next day. (Monthly Jour. Med. Science, Sept. 1846, p. 228.) The exact quantity taken in this case is not known; but there is no doubt that the poison is but little inferior to arsenious acid in activity, and the symptoms and appearances after death from a fatal dose would be similar. This substance is not much known in England. A woman was convicted in France for poisoning her husband with it in 1844. (Briand, Man. Comp. de Méd. Lég. 228.) It owes its poisonous properties to arsenious acid, of which, with the metal, it appears to be a mechanical mixture.

According to Dr. Schütte, it contains, as it is usually sold, from four to eleven per cent. of white arsenic. This gentleman has reported one case in which it was homicidally administered by a man to his wife. The prisoner, Dombrowsky, was tried before the Court of Wolfenbüttel, in July, 1853, convicted of murder by poison, and subsequently executed. The quantity of the powder taken by deceased is unknown. The symptoms from which she suffered were violent vomiting and purging, severe pain in the abdomen, and great thirst. She died in six days. The principal appearances were softening and excoriation of the mucous membrane of the stomach, striped or striated inflammatory redness of the mucous membrane, with bloody points about the cardia. The intestines were also inflamed. Small, black, metallic-looking particles were found in the contents of the stomach; and these, on analysis, proved to be arsenic. The quantity found amounted to about nineteen grains. Some of the same kind of powder was taken from the prisoner's pocket. (Casper's Vierteljahrschrift, 1854, ii, p. 230; and Otto, Ausmit. der Gifte, 1856, p. 56.)

This metallic-looking powder forms what may be called *black* arsenic. Dr. Chevers refers to a case that occurred in India, in which some difficulty arose in respect to the evidence, in consequence of a witness describing an arsenical powder as black. (Med. Jur. for India, p. 74.) It was thought that the witness had made a mistake in the description; but the arsenic was probably in the form of fly-powder. It would be known from white arsenic colored by soot, by the great weight and metallic appearance of the black particles.

Analysis.—When boiled in water, arsenious acid is dissolved, and the appropriate tests may be then applied to the solution. When a small portion is gently heated in a reduction-tube, a ring of arsenious acid is obtained as well as a ring of metallic arsenic. With soda-flux, a well-defined metallic crust is procured, possessing the characters already described (ante, p. 348). This compound is used for destroying flies as well as vermin.

FLY-WATER is a name applied to solutions of arsenic and of various arsenical compounds in water. Mixtures of this kind may be formed by dissolving one part of the arsenite of soda or potash, and two parts of sugar, in twenty parts of water. Paper soaked in this solution, and dried, is used for poisoning flies; and perhaps this is the safest form in which arsenic can be used for such a purpose.

A case of poisoning by fly-water, in which two grains and a half of arsenious acid destroyed the life of an adult in thirty-six hours, will be found reported in the Medical Gazette (vol. xxxix, p. 116).

ARSENIC ACID.

Symptoms.—This is an artificial product almost entirely confined to the chemical laboratory. Orfila states that it is a more powerful poison than arsenious acid, but he does not adduce any cases in support of his opinion. I have not been able to find any case of poisoning by it in the human subject. Dr. Glover ascertained that four grains of the acid, dissolved in two drachms of water, and introduced into the stomach of a stout rabbit, killed the animal in four hours, with the symptoms of irritant poisoning, and an affection of the nervous system. (Ed. Med. and Surg. Jour. vol. lviii, p. 121.)

Treatment.—The hydrated oxide or acetate of iron would be more likely to act as an antidote in poisoning by arsenic acid, owing to the great solubility of this compound and its tendency to combine with the oxide.

Analysis.—Arsenic acid is a white uncrystalline deliquescent solid. 1. It is not entirely volatilized on platina foil by the flame of a lamp. 2. It is very soluble in water, forming a highly acid solution. 3. It is precipitated of a brick-red color by nitrate or the ammonio-nitrate of silver. In these characters it differs from arsenious acid. 4. It yields readily an arsenical sublimate with charcoal. 5. It yields deposits with copper and muriatic acid, as in Marsh's apparatus. But Dr. Rainey has shown that Reinsch's process does not act so delicately with the arsenic, as with arsenious acid. The arsenic may, however, be converted to arsenious acid by mixing it with sulphurous acid and evaporating the liquid to dryness. Arsenic acid is also precipitated of a pale-yellow color, although slowly, by sulphuretted hydrogen gas. In these properties it resembles arsenious acid.

ARSENIATES OF POTASH AND SODA.

Symptoms and appearances.—The arseniates of potash and soda must be regarded as active poisons, although there are but few instances on record in which life has been destroyed by them. Dr. Christison states that, in the course of his reading, he has met with only two reported cases of poisoning by arseniate of potash. (Op. cit. 284.) M. Bouley administered this salt to seven horses, from the effects of which they all died. On inspection, it was observed that there was well-marked inflammation of the stomach, intestines, and bladder, and there were ecchymoses in the left ventricle of the heart. The contents of the viscera in one horse yielded no traces of arsenic—a fact probably to be ascribed to the violent diarrhœa from which the animal had suffered. (Orfila, Toxicologie, i. 452.) An attempt at murder by the arseniate of potash was the subject of a trial in France in 1844. This poisonous salt had been maliciously put into a bottle of wine. The prosecutor swallowed a mouthful, and, from finding the liquid very bitter, he spat out the greater portion. His wife also tasted it, but drank only a small quantity. In the course of the night the prosecutor was seized with severe colic, vomiting, general prostration of strength, and stupor; the wife suffered from similar symptoms. The medical man who had been called to them, finding that but a small quantity of wine had been taken from the bottle, referred the symptoms to indigestion. The next morning the prosecutor gave a portion of the suspected wine to a dog: the animal suffered from violent vomiting and convulsions, and died in four hours. The wine was analyzed by M. Chevallier, and found to contain about one drachm of arseniate of potash to a pint. A person, in whose possession a large quantity of arseniate of potash was found, was tried for this nefarious attempt to poison; but he was acquitted. (Journal de Chimie Médicale, 1854, p. 524.) A coarse sort of blotting-paper, soaked in a solution of arseniate of potash, is now extensively sold under the name of "*Papier Moure.*" It is erroneously represented that the substance with which it is impregnated is not poisonous to human beings.

Two cases are reported of poisoning by arseniate of soda. Two young men sent to a druggist's for doses of tartrate of soda, in place of which arseniate of soda was sent by mistake. In five minutes after the substance had been taken, they were attacked with violent cramps in the stomach. One died, and the other remained for some time in a dangerous condition. (Amer. Jour. Med. Sci. Oct. 1852, p. 553; and Wharton and Stillé's Med. Jur. p. 454.)

Treatment.—A solution of an arseniate is copiously precipitated by the hydrated oxide, or a solution of acetate of iron: hence these substances might be administered with a fair prospect of benefit.

Analysis.—Arseniate of potash is a white deliquescent substance, fixed when heated, and very soluble in water. The same tests may be applied to it as to ARSENIC ACID. Marsh's process acts with much more certainty and delicacy than that of Reinsch (ante, p. 353). In order to separate the whole of the arsenic, the liquid may be acidulated with muriatic acid, and a current of sulphuretted hydrogen gas passed into it.

The BINARSENIATE OF POTASH is known under the name of *Macquer's neutral arsenical salt.* The liquid known as *Pearson's* solution, which is still used medicinally in France, is a mixture of one grain of *arseniate of soda* to one ounce of distilled water.

SULPHURETS OF ARSENIC.

There are several kinds met with in commerce—ORPIMENT or YELLOW ARSENIC, and REALGAR or RED ARSENIC. They are poisonous in consequence of their containing a large proportion of free arsenious acid; this sometimes amounts to as much as 30 per cent. of their weight. They are occasionally used as poisons: in several criminal trials in England it has been proved that orpiment was the substance employed. Orpiment and realgar are employed in the arts, and are procurable by artisans with the most destructive facility. On one occasion, a quantity of red powder, brought to me by a mechanic as iron-rust, which he was carrying loosely in his waistcoat pocket, turned out to be realgar! From the brilliant colours of these compounds, they are used in painting, dyeing, paper-staining, and even in the coloring of toys and sweetmeats for children! It is remarkable that, under these circumstances, accidents are not more frequent. (See Ann. d'Hyg. 1848, p. 358.)

It is in the state of yellow sulphuret that arsenic is so commonly found in the stomach after death when the body has been buried for a long period. This arises from the action of sulphuretted hydrogen, generated by decomposition, on the white arsenic taken during life. In some instances, the coats of the stomach and intestines may present deep yellow patches from this change taking place in the tissues. This conversion of white to yellow arsenic, is, in general, only partial (ante, p. 182).

Symptoms and appearances.—The sulphurets of arsenic produce symptoms and appearances after death similar to those caused by arsenious acid; but the dose required to destroy life must vary according to the proportion of arsenious acid with which the sulphuret happens to be mixed. This is not a common form of poisoning; the intense colour of the poison would lead to suspicion. It was with orpiment that Mrs. Smith was poisoned at Bristol in 1835. (Med. Quart. Rev. July, 1835, p. 390.) This poison, from its colour, may be given or taken, by mistake, for mustard or turmeric. In a case which occurred to Dr. Jochner, two persons partook of some porridge, in which orpiment had been put, by mistake, for turmeric. They suffered from continual vomiting, burning pain in the stomach, and collapse. One, an old man, died in twenty-two hours; the other, a boy, recovered. Evidence of violent inflammation was found in the gullet and stomach, the mucous coat of the latter being softened and thickened. There was a sphacelated spot, one inch in diameter, in the œsophagus; and another in the stomach, three inches in extent. (Wharton and Stille, Med. Jur. 434.) According to Dr. Chevers (Med. Jur. for India, p. 74), orpiment is much used in India both as a medicine and as a poison. He refers to eight instances in which this poison was found, either in food, or in the stomachs of persons who had died under symptoms of irritant poisoning. The sulphuret was detected in the remains of two bodies after ten months' interment without coffins. Orpiment and realgar are sold openly in India, and are used as depilatories. Orpiment has been known to cause death by *external* application as a depilatory (see Annales d'Hygiène, 1834, 459); a result which might be expected from the quantity of arsenious acid with which it is mixed. There is a form of depilatory used, which consists of one part of orpiment, twelve parts of quicklime, and ten parts of starch, made into a soft paste with water (Pereira, i, 162), the use of which must always be attended with danger.

Treatment.—The promotion of vomiting with the exhibition of mucilaginous liquids can alone be trusted to.

Analysis.—The powdered sulphurets yield a solution of arsenious acid on boiling them in water acidulated with muriatic acid. They readily give the well-known sublimates of metallic arsenic, both with soda-flux, silver, and in the hydrogen apparatus. They also yield a deposit of arsenic when boiled with copper and muriatic acid. Orpiment is insoluble in muriatic acid, but it is readily dissolved by a solution of potash. *Organic mixtures.*—The sulphuret being insoluble in water, it is in general easily separated mechanically by allowing the matters mixed with it to become dry upon bibulous paper. If the sulphuret cannot be separated mechanically, the organic matter suspected to contain it should be dried and boiled with nitric acid to dryness, until it is destroyed. Any sulphuret will be then found, under the form of arsenic acid, soluble in water. Another impure sulphuret, sold as *King's yellow*, is composed, according to Dr. Christison, of sulphuret of arsenic, lime, and sulphur. It is highly poisonous, and is extensively sold as a pigment. A case of alleged poisoning by this substance has been already related (ante, p.

209.) The patient died of inflammation of the air-passages. The sulphuret of arsenic is easily separated from the pigment by digestion in caustic alkali.

SOLUTION OF CHLORIDE OF ARSENIC. (LIQUOR ARSENICI CHLORIDI.)

This is a pharmacopœial solution of arsenic in diluted hydrochloric acid. It contains one grain and a half of arsenious acid in one fluid-ounce, which is equal to the small proportion of three-sixteenths of a grain to a fluid-drachm. Mr. Phillips states that it is a highly poisonous preparation, and from a case which I saw in Guy's Hospital in May, 1857, this statement is confirmed. An adult female took, in three doses, thirty minims over a period of twenty-four hours. The quantity of arsenic thus taken was not more than the *tenth part* of a *grain*, and yet the symptoms which followed were of a severe kind, resembling those of chronic poisoning. There were constriction of the throat, pain and irritation of the stomach and bowels, tingling and numbness of the hands and feet, loss of muscular power, and a feeling of extreme depression. The medicine was withdrawn, and the patient slowly recovered. It seems that she had not taken arsenic previously, and there was no evidence of the existence of a peculiar susceptibility to the effects of this poison. The quantity taken was very small to produce such alarming effects. The usual medicinal dose of this solution is from three to ten minims. It has about three-eighths of the strength of the solution of arseniate of potash.

ARSENURETTED HYDROGEN.

This is a gaseous poison of arsenic, producing, when respired in small quantity, very serious effects upon the system. It has already occasioned death in at least three instances. The gas is an artificial product, and is formed in a chemical laboratory in various ways,—one method has already been described in speaking of Marsh's process (ante, p. 353): and its highly poisonous properties render it necessary that caution should be used in the employment of this mode of testing. The gas is most effectually decomposed, and prevented from diffusing itself, by passing it into a solution of nitrate of silver, or chloride of gold. (See ante, p. 354.) This form of gaseous arsenical poisoning has been hitherto purely accidental. It is stated that Gehlen, a German chemist, was killed by accidentally breathing a small quantity. Suspecting that the gas was escaping from some part of the apparatus he was using, he applied his nose for the purpose of detecting it; and although he respired but a small quantity, probably a few hundredths of a grain of arsenic only, he was seized in about an hour afterwards with vomiting, shivering, and great prostration of strength. He died on the ninth day. The most complete history of this kind of poisoning has been published by Dr. O'Reilly, of Dublin. I am indebted to him for the particulars of the case.

Symptoms and appearances.—A gentleman, for the sake of experiment, wished to respire about one hundred and fifty cubic inches of pure hydrogen gas. It unfortunately happened that the sulphuric acid, which he used for making the hydrogen, was largely contaminated with arsenic. Immediately after he had respired the gas, he was seized with giddiness and fainting, constant vomiting of a greenish-colored matter, and dull pain in the region of the stomach. There was also complete suppression of urine. He died in about six days. On dissection, the liver and kidneys were found of a deep indigo colour,—the mucous membrane of the stomach was easily separated; and there were two distinct patches of inflammation at the greater curvature. There was a quantity of reddish-colored fluid effused in the chest, in about ten ounces of which Dr. O'Reilly detected arsenic by the use of Marsh's process. From experiments made subsequently on the sulphuric acid, it is supposed that the deceased must have inhaled a quantity of arsenic equivalent to about twelve grains of arsenious acid. A case of poisoning by this gas occurred in England in December, 1836. A young gentleman was killed by respiring the gas, evolved from a mixture of arsenic, zinc, and sulphuric acid. Death did not take place until twenty-four days after the accident. It appears that in this instance but a very small portion could have entered into the lungs. I am indebted to Dr. Mouatt, of Calcutta, for the particulars of another case,—that of Prof. Robertson, of Calcutta Medical College, who, while delivering a lecture on arsenic, accidentally breathed a portion of this gas which was escaping from a Marsh's apparatus. The first symptoms were a sense of burning and of constriction in the throat, followed by irritability of the stomach, vomiting of liquid, at first bilious, and afterwards coffee-colored, with a burning pain through the whole alimentary canal. Four pints of bloody urine were passed, and this, on examination, was found to contain arsenic. There was constipation of the bowels, with fever, a full, hard, frequent pulse, dry, hot, unperspiring skin, restlessness, anxiety, and great prostration of strength. He did not recover from these symptoms until the twenty-second day.

The detection of arsenic in the serous liquid of the chest proves that this poison is eliminated not merely by the natural secretions, but also in the morbid effusions. Dr. Chatin has recently proposed this as an additional means of diagnosis in poisoning by arsenic. A blister was applied to the chest of a female laboring under the effects of this poison. About ten drachms of the serum were collected, and this quantity contained sufficient arsenic to give sixteen well-marked metallic deposits by the use of Marsh's apparatus. (Jour. de Chimie, 1847, 329.) *Analysis.*—The chemical properties of this gas have been already described. (See Marsh's process, ante, p. 353.) It is colorless, possessed of a disagreeable odor resembling that of garlic, inflammable, burning with a bluish-white flame, and evolving an abundance of white solid vapor. While burning, it is converted into water and arsenious acid. On cold surfaces it deposits metallic arsenic, suboxide of arsenic, arsenious acid, and water. (For the characters of the deposits, see p. 355.) It is decomposed by chlorine, forming muriatic acid and chloride of arsenic; also by those metallic solutions the metals of which have a weak affinity for oxygen. Paper soaked in a solution of nitrate of silver and held over the gas, is immediately blackened. If it be passed into the solution, the silver is reduced, and arsenic acid is dissolved.. The gas is not soluble in water, and the air of water decomposes it. At a red heat the metal is entirely deposited, and hydrogen escapes. This is applied as an adjunct test in Marsh's process. It is known from most other gases in being totally absorbed by a solution of sulphate of copper (Gregory). The specific gravity of the gas is 2·695. It contains by weight 96·2 per cent. of arsenic; and as 100 cubic inches would weigh 82·17 grains, every cubic inch will contain more than 8-10ths of a grain of arsenic in a finely-divided or gaseous state! It is therefore one of the most formidable poisons with which we are acquainted. No treatment can save life when it has been once respired.

CHAPTER XXVI.

LIQUID MERCURY NOT POISONOUS—CORROSIVE SUBLIMATE—TASTE AND SOLUBILITY—SYMPTOMS—ITS EFFECTS COMPARED WITH THOSE OF ARSENIC. SLOW OR CHRONIC POISONING—ACTION OF MERCURY IN VAPOR—SALIVATION FROM SMALL DOSES OF MERCURIAL MEDICINES—FROM OTHER CAUSES—CANCRUM ORIS—EFFECTS OF EXTERNAL APPLICATION—APPEARANCES AFTER DEATH—FATAL DOSE—PERIOD AT WHICH DEATH TAKES PLACE—TREATMENT.

General remarks.—METALLIC MERCURY, in a state of liquid, appears to be entirely destitute of poisonous properties. Numerous cases are recorded in which this substance has been swallowed with impunity. One was the subject of a report to the Westminster Medical Society, in November, 1842. The individual in this case labored under obstinate constipation which ended fatally. Five days before death, half a pound of liquid mercury had been swallowed as a remedial agent, and no ill effects followed. On an inspection of the body the mercury had wholly disappeared. Much larger quantities of the metal have been taken without injurious consequences. In a case of obstinate constipation, after trying, without effect, all the common purgatives, Dr. Borgstedt prescribed for a female, æt. 42, two pounds of mercury, to be swallowed at intervals! The metal remained nine days in the body, and was perceptible to the feel through the abdomen. The last portions of metal were passed by stool on the fourteenth day. Only five-sixths of the quantity administered were recovered. Slight salivation appeared about this time, but this after-effect was speedily subdued. (Casper's Wochenschrift, April 12, 1845, 249.) In the same journal, Dr. Kerstein relates a somewhat similar case, in which, under an attack of ileus (ante, p. 126), he gave to a man, æt. 28, two pounds of quicksilver, in four doses,—six ounces at each dose. Croton oil was then prescribed, and after eight days the bowels were moved, the greater part of the metal being passed unchanged, excepting some portion which had been converted into black oxide (30 Mai, 1846, 355). Many similar cases might be quoted, all tending to show that metallic mercury is inert.

The most important mercurial poison is CORROSIVE SUBLIMATE; there are some other preparations of the metal, which will require a brief notice.

CORROSIVE SUBLIMATE.

This substance is commonly seen under the form either of very heavy crystalline masses, or of a white powder, and is known by the chemical name of *Chloride*, *Perchloride*, or *Bichloride of Mercury*. The term *Chloride*, has been, and is now by many chemists also assigned to calomel. To prevent any confusion from scientific chemical nomenclature, the old and popular name of corrosive sublimate is retained. This compound is not often taken as a poison. In the coroner's report for 1837–8, there were about fifteen fatal cases of mercurial poisoning, in twelve of which corrosive sublimate was the poison taken. It is chiefly used for the purposes of a bug-poison, in preserving timber from the dry-rot, and in bronzing gun-barrels. It is freely retailed to the public at the rate of twopence for from one to two drachms; if exceeding this quantity, the price is sixpence per ounce. This may guide the witness when he has to judge of the quantity taken, by the price paid.

Taste and solubility.—The *taste* of corrosive sublimate is powerfully acrid and metallic, so that no poisonous quantity of it can be easily swallowed without the person becoming immediately aware of the fact. It is very *soluble* in water, hot or cold, and speedily sinks in it, in which properties it differs strikingly from arsenic. I have found, by experiment, that one hundred grains of a cold saturated solution hold dissolved, at a maximum, ten grains of corrosive sublimate; and it is stated by most chemists that two parts of boiling water (212°) will dissolve one part of the poison. The Pharmacopœial solution (*Liquor Hydrargyri Bichloridi*) used in medicine, contains only half a grain of corrosive sublimate to one fluid-ounce. Half a grain of hydrochlorate of ammonia is added. Corrosive sublimate is also readily dissolved by alcohol and ether; the last body takes up one-third of its weight, and has the property of extracting it from its aqueous solution,—a principle which is sometimes advantageously resorted to for separating the poison when dissolved in organic liquids. It is soluble, without change, in nitric and muriatic acids. The solubility of this poison in an alcoholic liquid (whiskey) was a material question in Reg. v. Walsh (Kilkenny Summer Assizes, 1850). The prisoner had poured the whiskey on the poison in a cup, and the deceased had drunk it. It produced its usual effects. Some testimony was brought to show that the spirit would not dissolve enough to destroy life, but this was confuted. The prisoner had wilfully given some of the whiskey shortly before to another man, who did not perceive any taste, and did not suffer any injurious effects, while the deceased complained that it had a "queer, burning taste," and that he felt a burning in his mouth and throat. The whiskey was not, in fact, poisoned, but it only became so when poured on the corrosive sublimate in the cup! This gave some plausibility to the defence. (Med. Gaz. 1850, vol. xlvi, p. 253.)

SYMPTOMS.—ACUTE POISONING.—The symptoms produced by corrosive sublimate, generally come on immediately or within a few minutes after the poison has been swallowed. It differs from arsenic in producing immediately a chemical or corrosive action on the animal membranes. There is perceived a strong metallic taste in the mouth, often described as a coppery taste, and there is, during the act of swallowing, a sense of constriction almost amounting to choking or suffocation, and a burning heat in the throat, extending downwards to the stomach. In a few minutes violent pain is felt in the abdomen, especially in the region of the stomach, which is increased by pressure. Pain in the abdomen is, however, sometimes wholly absent. There is nausea, with frequent vomiting of long stringy masses of white mucus, mixed with blood; and this is accompanied by profuse purging, the evacuations being of a mucous character and in some cases marked or streaked with blood. The countenance is sometimes swollen and flushed, in other cases it has been pale and anxious. The pulse is small, frequent, and irregular, becoming scarcely perceptible as the symptoms progress. The tongue is white and shrivelled,—the skin is cold and clammy, the respiration difficult; and death is commonly preceded by syncope, convulsions, or general insensibility. The internal parts of the mouth, with the lips, are white and swollen, and have presented an appearance as if the cavity had been washed with a solution of nitrate of silver. It is worthy of remark that on a few occasions the tongue and mouth have not presented these changes. Suppression of urine has been frequently noticed among the symptoms. It existed in a well-marked case of poisoning by this substance at Guy's Hospital. The patient lived four days, and did not pass any urine during the whole of this time (p. 401, post). This symptom was also observed in a case reported by Dr. Wegeler (Casper's Wochenschrift, Jan. 10, 1846, p. 30), in which a youth, æt. 17, swallowed three drachms of the poison, and died on the sixth day. During the last three days, no urine was secreted. The case was otherwise remarkable from the fact, that no pain was experienced on pressure of the abdomen, and that the pulse underwent no change until shortly before death. In another, reported by Dr. Herapath, in which a scruple of corrosive sublimate in solution was swallowed, suppression of urine and salivation came on on the third day, and the patient died on the ninth day. (Lancet, Dec. 13 and 27, 1845, pp. 650, 698.) In a case observed by Mr. Morris, the quantity of urine secreted was small, and it produced a scalding pain when voided. (Prov. Med. Journ. Nov. 18, 1843, p. 126.) In this instance there was no purging.

As contrasted with the effects of arsenic, it may be observed, 1, that corrosive sublimate has a well-marked taste; 2, it produces violent symptoms within a few minutes; and 3, the discharges are more frequently mixed with blood. The symptoms produced by corrosive sublimate, in the first instance, resemble those of cholera; if the person should survive several days, they, in some respects, assume the character of dysentery,—tenesmus and mucous discharges mixed with blood being very frequently observed.

A swelling of the salivary glands and an increased flow of saliva are commonly enumerated among the symptoms, but these are by no means necessary attendants on cases of acute poisoning. Unless the patient survive two or three days, salivation is not commonly observed among the symptoms, and even in this case it is not often met with. In a case which occurred to Dr. Venables, in which two drachms of the poison had been taken, and the woman survived eight days, there was no salivation. In another, reported by Mr. Wood (Ed. Med. and Sur. Jour. li, 141), in which half a teaspoonful of the poison was taken, salivation was profuse in the course of a few hours. In a case which occurred at Guy's Hospital, in February, 1843, in which two drachms had been taken, salivation commenced in four hours: but this is by no means the earliest period. Dr. Percy relates a case in which the saliva was flowing profusely an hour and a half after the woman had taken a dose of thirty grains. (See Med. Gaz. 1848, i, 942.) In these cases of early salivation, it is alleged that fœtor of the breath is absent, and that the salivation is the result not of absorption, but of a local irritant effect exerted by the corrosive sublimate. But most practitioners will look merely for an effect on the salivary organs. The local action of the poison is, in some instances, sufficient to account for the abundant flow of saliva independently of the influence of absorption. In a case, in which half a drachm of the poison in powder was placed by a woman on her tongue, the saliva flowed abundantly from the mouth, and the lips were much swollen. (Prov. Med. Jour. Nov. 18, 1848, p. 127.) This was undoubtedly due to a local effect of the poison.

The following cases will illustrate the ordinary effects of this poison. J. W., an adult, æt. 38, who an hour before had masticated and swallowed about two drachms of corrosive sublimate in coarse lumps, was admitted into Guy's Hospital, in February, 1843. There was great depression of the system; the limbs were quite cold; the respiration was natural; the pulse small, wiry, and scarcely perceptible; the tongue swollen, as well as the lips. The man was sensible; and complained of constriction of the throat. Albumen was immediately administered to him. In three hours after his admission, the lips were swollen and tender, as well as the gums; there was incipient salivation, with pain extending down the gullet, into the stomach. The act of swallowing gave rise to much pain. The man had vomited several times, and brought up a quantity of a yellow-colored matter, interspersed with blood; he complained of slight pain in the abdomen. There were spasms of the lower limbs; the knees were drawn up; the pulse was small and scarcely perceptible; the tongue was white, and so much swollen that he could not protrude it from his mouth. These symptoms continued, more or less, during the four days which he survived. There was complete suppression of urine, with occasional discharges of mucus mixed with blood, from the bowels. Before death, there was delirium; the man died in a comatose state. (Guy's Hosp. Reports, April, 1844, p. 24.)

In 1846, a woman, æt. 25, was admitted into the hospital laboring under symptoms of poisoning by corrosive sublimate, which she had taken dissolved in spirits of camphor. She had put into her mouth about a tablespoonful, but her husband seized her by the throat and prevented her from swallowing more than a very small portion of it; she spat the greater part out again. She immediately felt a strong coppery taste, with burning heat in the mouth and throat, attended with difficulty in swallowing. In about five or ten minutes she vomited. When admitted, there was no collapse or coldness of the skin or limbs; on the contrary, they were warm and moist. Her face was much disfigured by the projection of the lips, which were considerably swollen and very tender. On examination, the throat was not found to be much injured, —the action of the poison having been chiefly confined to the mouth. Some of the fluid had run down from each corner of the mouth, producing patches of redness externally; the mucous membrane of the lips and tongue had a whitened aspect; there was retching, as well as vomiting, but not violent. The vomited matter was but slightly tinged with blood. The pupils were dilated, and the conjunctiva inflamed. There was painful headache; the pulse, which was at first unaffected, subsequently increased to 112, and became irregular; the breathing was unimpaired. There was no purging, nor any *pain* at the stomach. There was great thirst, with occasionally a tendency to sleep. The pain in the throat afterwards extended to the chest; the submaxillary glands became enlarged and tender; but the gums were unaffected; and there was no salivation nor fœtor. In two days the symptoms had abated; the mucous membrane of the lips peeled off, and the throat was painful. After this, the woman gradually improved, but her mouth and gums remained tender for four days.

In April, 1846, a man swallowed about a drachm of corrosive sublimate mixed with half a pint of water and some hydrochloric acid. He experienced great heat and soreness of the throat and gullet in the act of swallowing the poison; and was shortly afterwards seized with violent vomiting and purging. He was brought to Guy's Hospital in half an hour, and he was then laboring under the following symptoms: There was coldness of the limbs, with a general lividity of the surface, especially about the backs of the hands, cheeks, and forehead, which presented a purplish-black appearance. He was affected with shivering and frequent chattering of the teeth, without any sense of cold in any part of the body; the pupils were at first natural; they were subsequently contracted, pulse about 100, small and feeble. There was violent vomiting as well as purging; the former occurring at first every two or three minutes, subsequently every half hour. The vomited matters consisted of mucus tinged with blood, mixed with the albumen which had been administered. There was purging every hour throughout the day: the discharges, which consisted at first of little else than mucus and blood, became more and more feculent. The tongue was covered with a grayish-white crust, resembling the effects of nitrate of silver; its margin was of a bluish color. There was great heat, with soreness of the mouth, throat, and gullet, especially during the act of swallowing any liquid; the gums had receded from the base of the crowns of the teeth, and presented a leaden-blue margin: the abdomen was generally free from pain; there were occasional cramps of the lower limbs. On the second day there was a remission of the symptoms, but the vomiting and purging returned; there was delirium with tremor and cramps of the limbs. He died on the fourth day. There was no salivation.

As in the case of arsenic, the symptoms caused by this poison are liable to great variation, even when the dose is the same. In March, 1848, a young man took, on an empty stomach, a drachm of corrosive sublimate partially dissolved. When seen some hours afterwards, his face was pale,—the surface of the body cold, especially the limbs. Vomiting had occurred in a few minutes after he had taken the poison, and had continued almost incessantly,—the fluid ejected consisting of a frothy liquid mixed with blood. There was pain in the stomach, but this was not increased by pressure. No injury of the mucous membrane of any part of the mouth could be detected. This is an important fact in reference to the recognition of such cases, since, as in poisoning by oil of vitriol, the mouth may present no marks of a chemical action. On the second day, the pain continued with the same kind of vomiting,—the pulse was small and weak, with intermissions; the tongue red and dry. On the third day there was a remission of the symptoms; the vomiting and purging had ceased, and there was an abatement of the pain. On the fourth day he died,—the symptoms having become worse; there was purging of a bloody liquid, and great difficulty of breathing, with blueness of the lips from obstruction of the bronchial tubes by mucus. No urine was passed during the last twenty-four hours; the intellect was not affected at any time. (Mr. Wade, in Med. Gaz. 1848, vol. xli, p. 779.)

In a case which was the subject of a criminal trial, in which an unknown quantity of this poison was given in whiskey, the symptoms were of a very marked kind; there was a burning pain in the mouth and throat, with immediate sickness; pain in the stomach, as well as about the mouth and head; the vomiting was incessant. There was profuse salivation on the third day, with a discharge of shreds of corroded membrane mixed occasionally with blood. The breath was offensive; the tongue swollen and protruding; the teeth were black; the gums and palate ulcerated; the salivation continued, the man became weaker, and before death, on the fifteenth day, there was a discharge of blood from his mouth. (Med. Gaz. 1850, vol. xlvi, p. 254.)

Chronic poisoning.—The symptoms are much modified when the poison is taken in small doses at certain intervals for some days or weeks. There are colicky pains, with nausea, vomiting, general uneasiness, and depression. The salivary glands then become painful, inflamed, and ulcerated; the tongue and gums are red and swollen, sometimes ulcerated, and there is a peculiarly offensive smell (fœtor) of the breath. A deep blue line, like that observed in poisoning by lead, is sometimes found at the edges of the gums where they join the teeth. The patient experiences difficulty of swallowing and breathing. The constitutional effects are indicated by looseness of the bowels, spitting of blood, cough, general trembling of the limbs and palsy, with slow fever and emaciation, under which the patient sinks.

Should the person survive some time, salivation is commonly met with; but in looking for this as a special and characteristic symptom of mercurial poisoning, a medical jurist must remember, that some persons are wholly unsusceptible of this condition. On the other hand, there are cases in which the salivary glands are easily excited, so that the usual innocent doses of mercurial medicines have been known to produce salivation to such a degree as to cause death. Facts of this kind are of some importance, since charges of malapraxis may be easily raised in respect to them. Dr. Christison mentions a case in which two grains of calomel destroyed life by the severe salivation induced, as well as by ulceration of the throat. Another was mentioned to me by a pupil, in 1839, in which five grains of calomel killed an adult by producing fatal salivation. From some cases related by Mr. Samuel, of Newark, it appears that two grains of calomel, divided into three powders, were given in the proportion of one powder daily (two-thirds of a grain), to a little boy aged eight. This small dose produced violent salivation, sloughing and disease of the jaws, from which he was some weeks in recovering. In another instance, a girl, aged five, took daily, for three days, three grains of mercury and chalk-powder. Her mouth was severely affected, sloughing ensued, and she died in eight days. In a third case, a boy, æt. 11, took three doses of this powder, one of six grains on the 14th, a similar dose on the 17th, and four grains on the 20th, making altogether, sixteen grains in a week. Profuse salivation followed, sloughing commenced in both cheeks, and rapidly extended through them. The boy died in four days. Previously to taking the mercury, the boy had recovered from an attack of fever. (Lancet, Dec. 20th, 1851, p. 579.) In a fourth case, three grains of blue pill, given twice a day for three days, making eighteen grains, were ordered for a girl aged nineteen, who complained of a slight pain in her abdomen. Severe salivation supervened, the teeth separated, and she died in twelve days. With respect to the effects of corrosive sublimate, Dr. Christison states that he has known three grains only of this substance, in three doses, cause violent salivation. (Op. cit. 408.) When this state results from the use of mild mercurial medicines in small doses, the severe effects may be referred to idiosyncrasy, or a state called intolerance (ante, p. 101). A person may die under these circumstances,—either from simple exhaustion, or from excessive sloughing of the throat with disease of the bones. When an individual has recovered from the first effects of acute poisoning by corrosive sublimate, he may die at almost any period from these secondary consequences.

It is admitted by toxicologists, that salivation may be intermittent, *i. e.* that it may cease and reappear without more mercurial poison, or any mercurial preparation, being given in the interim, although such cases are rare. As a matter of medical jurisprudence, this important question was brought to an issue at the trial of Butterfield, at Croydon, in 1775. The deceased was supposed to have been killed by the administration of small doses of corrosive sublimate, and the fact of his having been salivated at or about the time of the alleged administration, was regarded as a proof of poisoning. In the defence it was urged, that the deceased had been salivated two months previously, under a common mercurial course,—and although the salivation had ceased for that period, it was probable that this was nothing more than a recurrence of the former:—it did not prove that there had been any fresh administration of mercury in the interim. There was a difference of opinion on this point among the witnesses, as there probably would be in the present day, if each relied upon his own personal experience. However, one of the witnesses stated that he had known salivation to recur without a fresh exhibition of mercury after the long interval of *three months*, and the prisoner was acquitted. Cases are reported of salivation recurring after intervals even longer than this. One is quoted by Mr. Swan, in which salivation recurred after an interval of six months. (On the Action of Mercury, 1847, p. 4.)

It is necessary to bear in mind that salivation is not necessarily connected with the administration of mercury, and therefore, when taken alone, it can never furnish evidence of mercurial poisoning. Salivation may come on *spontaneously* from disease in the salivary organs; or it may arise from simple mechanical causes. Dr. Mulock has communicated a case to the Dublin Hospital Gazette, in which profuse salivation was occasioned by the introduction of a set of artificial teeth. (Sept. 15, 1845, p. 35.) It may be produced by many other substances besides the preparations of mercury. Thus it has been known to follow the use of the preparations of gold, copper, bismuth, lead, antimony, iodine, iodide of potassium, croton oil, opium, prussic acid, sulphuric acid, arsenic, colchicum, foxglove, and cantharides. Some have asserted that an offensive odor of the breath, a brassy taste in the mouth, and spongy and ulcerated gums, will indicate the salivation caused by mercury: but these characters have been equally met with in the salivation produced by arsenic and bismuth. (Prov. Med. Journ. Oct. 22, 1845, p. 638.) A case in which this question was material has been reported by Mr. Harding. (See Lancet, June 13, 1846, p. 654.) It appears from the researches of pathologists, that salivation is not so readily induced by mercurial preparations in young persons as in adults. Dr. Beck says that, in his experience, only one instance has occurred, in which a child, two years old, was salivated by five grains of calomel given in three doses in twelve hours: in two days the usual symptoms appeared. The child had been laboring under hooping-cough for several weeks, and was a good deal reduced. For other instances, see page 403, ante; but notwithstanding these cases, it is true as a general principle that young subjects are salivated with great difficulty, and at the same time the effects of mercury upon them are frequently more energetic and uncertain than they are in the adult. Hence mercurial preparations should be administered with great caution, when the strength of a child has been reduced by disease. In this state of constitutional depression, a single medicinal dose of calomel may sometimes prove fatal. (Dubl. Med. Press, May 12, 1847, p. 296; also, Amer. Journ. Med. Sciences, April, 1847, p. 509.)

In addition to the facts already detailed, respecting death from excessive salivation under the use of small doses of mercury, there are certain *morbid* states of the body which appear to have the effect of increasing the action of this medicine on the salivary glands. This kind of acquired idiosyncrasy exists especially in that form of disease called granular degeneration of the kidney, which is characterized in its early stage by albuminuria (see ante, p. 101). Dr. Craigie has observed, that when given to persons laboring under symptoms of granular kidney, a small quantity of mercury induces salivation, and renders the mouth tender and most painful. (Practice of Physic, ii, 1148.) Dr. Christison has repeatedly observed that mercurial action (salivation) is in these cases brought on by unusually small doses of the compounds of mercury, or unusually soon. A medico-legal case involving this question occurred at Reading in December, 1845. A man, laboring under disease of the kidneys, had placed himself in the hands of a person who promised to cure him. Part of the treatment consisted in the administration of small doses of mercury. Profuse salivation came on, and the patient, not finding himself relieved, applied to a medical practitioner. In about a fortnight afterwards he died, and a coroner's inquest was held, in order to determine whether he had not died from improper treatment. It appeared in evidence that some calomel pills were prescribed, and that had the prescription been followed, the deceased would have taken no more than six grains in the five days that he was under treatment; but, in consequence of some mistake, he took *eleven grains and a quarter*—i. e. *two grains and a quarter daily* for five days. On an inspection of the body, the gums were found ulcerated, and the mucous membrane of the tongue, mouth, and throat, was in a state of intense irritation. Both kidneys were enlarged, and in a diseased state. After hearing the evidence of several medical witnesses, the jury returned a verdict that deceased had died from natural causes.

In albuminuria, the kidneys appear after death larger than natural, of a dark or chocolate color, and they are evidently gorged with blood. During life, the disease is known, among other symptoms, by the presence of albumen in the urine. In granular degeneration the surface of the kidney is generally mottled or speckled: in the earlier periods of the disease the organ may be found larger than natural, and of softer consistence, while in the advanced stages it may be contracted and hard. The outer or cortical portion is commonly disorganized; it is granular, and of a pale yellow color. (Rees on the Blood and Urine, p. 154; and Gregory, Practice of Medicine, 691.)

Cancrum oris—Canker of the mouth.—Corrosive sublimate, as well as other mercurial preparations, is liable to produce gangrene of the mouth and throat, and thus destroy life. A disease called *canker*, or gangrene of the mouth, attended with ulceration of the gums and a falling out of the teeth, has been observed to occur in infants and children, to whom no calomel, nor any mercurial preparation whatever, has been given. Those who specially suffer from this disease are children badly fed and clothed, and generally laboring under, or recovering from, fever, small-pox, measles, or hooping-cough. It is, however, far more common as a consequence of measles than of other diseases, and it is always connected with a depressed state of the vital powers. Several cases of cancrum oris have been reported by Dr. Hennis Green. (See Lancet, Dec. 1839.) On these occasions, supposing any mercurial preparation to have been given medicinally, it may become a serious question whether death actually resulted from the mercury acting as a poison, or from the effects of disease. In some fatal cases that have occurred, the subject has become a matter of inquiry before coroners. Although salivation and its consequences,—ulceration of the gums and sloughing, are not common among children as an effect of mercurial preparations, yet it is clear, from the facts already cited (p. 403), that small doses of mercury may have occasionally a most violent effect upon them, and render the suspicion of poisoning probable. Of two children, whose deaths were the subject of investigation under these circumstances, one was affected with hooping-cough, and the other with measles. Powders containing calomel were prescribed in both cases—gangrene of the mouth supervened, and the children died. There was some reason to believe, from the evidence, that mercurial medicines had really produced the effect attributed to them, at least in one of the cases. In August, 1840, a charge was made against a medical practitioner of having caused the death of a child, aged four years, by administering an over-dose of some mercurial preparation. The child was laboring under hooping-cough, and some medicine was prescribed. On the fourth day the child complained of soreness of the mouth, the teeth became loose and fell out, the tongue and cheek were much swollen, and the child died in the course of a few days from gangrene in the left cheek. The answer to the charge was, that not a particle of mercury had been exhibited—a fact clearly proved by the production of the prescription-book of the medical attendant. This, then, was an instance in which gangrene from spontaneous causes, was mistaken for mercurial poisoning. Had the medicine prescribed contained any mercury, a verdict affecting the character of the practitioner would probably have been returned! (See also a case by Mr. Dunn, Med. Gaz. xxxiii, 57; and Br. and For. Med. Rev. October, 1844, p. 542.) Dr. Dugas considers that children between five and eight years of age are specially liable to this form of mercurial salivation. (Ed. Monthly Journal, May, 1851, p. 481.) It is worthy of remark, that in cases of this description, the popular opinion is generally supported by that of some medical practitioner,—showing how easily members of the profession, as well as the public, are led to refer the effects to what in many instances is only an *apparent* cause. An important case of this kind, in which the medical witness relied upon the "mercurial fœtor" as characteristic and distinctive, will be found in the Lancet (June 13, 1846, p. 654).

One form of chronic poisoning is seen in the effects produced by the *vapor of mercury* on persons engaged in certain trades. When mercury is respired in the state of fine vapor, it enters the body through the lungs, and is then capable of producing serious symptoms. Mercury may pass into vapor at all temperatures. In the trades in which the metal is used, it may thus penetrate into the system by slow degrees. The chronic effects are manifested by tremors and paralysis of the limbs—a state called shaking palsy—giddiness, loss of memory, disturbance of the intellectual faculties, salivation and ulceration of the gums, colic, general emaciation, and death. A blue line, as in chronic poisoning by lead, may be found at the edges of the gums. Water-gilders, and the manufacturers of looking-glasses, barometers, and thermometers, are subject to these disorders. The frequent contact of mercury with the hands may suffice to produce them in a modified degree. A case, in reference to the noxious effects of mercurial vapor, is reported by M. Chevallier. (Ann. d'Hyg. 1841, i, 389.) It was alleged that two children had suffered seriously in health in consequence of the distillation of mercury being carried on in an apartment below that in which they lived. They had general tremors and other symptoms indicative of mercurial action; but there was no salivation. It has been remarked that those who are subject to mercurial palsy are not very liable to become salivated. M. Chevallier detected mercury in the dust of the apartments on all the floors of the house; and his conclusion was, that the disordered health of the children was certainly due to these mercurial emanations. (See also Ann. d'Hyg. 1847, ii, p. 458.)

A remarkable instance of the noxious effects of mercurial vapor was observed in the case of the Triumph, while conveying a cargo of quicksilver, off Cadiz, in April, 1809. By some accident the leathern bags containing the metal burst, and *three tons* of quicksilver were dispersed through the vessel. The crew soon began to suffer from salivation, partial paralysis, and disorders of the bowels. In three weeks no fewer than 200 men were salivated. Two men died from excessive salivation; one lost his teeth, and his cheeks were in a gangrenous condition; the other lost the whole of his teeth, the greater part of his tongue, and at the time of his death the lower lip was in a state of gangrene. The interior of the ship was covered with a black powder, and the copper bolts were mercurialized. The vapor proved fatal to the animals on board; for nearly all the poultry, sheep, pigs, mice, goats, cats, and dogs, and even a canary bird, died from its influence. (Paris Med. Jur. ii, 461.) The poison in this case was not merely the vapor of metallic mercury, but probably in part oxide of mercury produced by friction.

Noxious effects may be apprehended when any operations with metallic mercury are carried on in small and ill-ventilated apartments, heated to a temperature above 70°. The best test for the detection of this vapor is the suspension of a piece of pure gold leaf in the apartment. If mercury be present, this will become slowly whitened by amalgamation.

Effects of external application.—Cases of poisoning by *external* application of corrosive sublimate are not very common. It acts through the unbroken skin, and more powerfully through ulcerated surfaces, producing severe local and constitutional symptoms, and even death. Two fatal cases of this kind have been reported by Mr. Ward, of Bodmin. (Med. Gaz. iii, 666.) A man, aged 24, rubbed over every part of his body one ounce of corrosive sublimate, mixed with six ounces of hog's lard, for the purpose of curing the itch. In an hour he experienced excruciating pain in the abdomen, and over the whole of his body; he said he felt roasted alive; he also suffered from intolerable thirst. The skin was found completely vesicated. He died on the eleventh day, having labored under bloody vomiting, purging, and tenesmus (straining). Salivation did not show itself until thirty-six hours after the application of the poison. The brother of the deceased, aged 19, rubbed in the same quantity of the poison. The symptoms were much the same, but more aggravated. There was constant vomiting, with complete suppression of the urine, and frequent bloody evacuations;—the salivation was not so severe. He died on the fifth day. On inspection, the stomach was found much inflamed, and partially ulcerated. The small intestines were also greatly inflamed throughout, and the lower portion of the colon and rectum were in a state of mortification. The bladder was contracted, and without urine. Thirty large worms were found alive in the stomach and intestines! (For another case, see Niemann, Taschenb. der Arzneiw. 452.) Mr. de' Ricci, of Ballymahon, met with two cases somewhat similar to those of Mr. Ward. Two boys, æt. 11 and 7 respectively, were treated by a quack for scalled head. This man, it appears, rubbed on the diseased surfaces an ointment consisting of two drachms of corrosive sublimate, and one ounce of tallow. It produced immediately the most intense suffering, and in from thirty to forty minutes there was vomiting, with pains in the bowels, followed by purging and bloody evacuations. The boys continued to get worse until death. The younger died on the seventh day, and the elder on the ninth. There was no salivation in either case: in the younger child there was an appearance like *cancrum oris* (ante, p. 405); in the elder, there was a rash like the eczema of arsenic about the mouth. In the younger child there was a complete suppression of urine, while in the elder it was merely diminished in quantity. On inspection, the morbid changes were chiefly confined to the stomach and bowels. The mucous membrane of the stomach was injected with red blood throughout; but there was no ulceration or softening. In one there were a few spots of effused black blood in addition to this injected appearance. In both, the intestines were highly inflamed and ulcerated; and in the younger, there were twenty-three intussusceptions (ante, p. 126). (Chemist, 1854, p. 760; and Dublin Quarterly Journal, Aug. 1854.) It is unfortunate that the stomachs were not submitted to analysis in these cases, to test the question of elimination. Death from the external application of corrosive sublimate has been the subject of a criminal trial. In this case, there were the usual symptoms of irritation, and the stomach and intestines were much inflamed. (Reg. v. Welch, Worcester Summer Assizes, 1845; Med. Gaz. xxxvi, 608.) The readiness with which this poison acts through the skin is proved by the following circumstance. M. Cloquet plunged his hands into a concentrated solution of corrosive sublimate, in order to remove some anatomical preparations. He did not wash his hands afterwards; and in about eight hours he was attacked with severe pain in the abdomen, constriction in the chest, painful respiration, thirst, nausea, and ineffectual attempts at vomiting. Under the use of diluents these symptoms were removed, but for eight days he suffered from pain in the stomach. (Galtier, i, 567.) There is reason to believe that, both in respect to themselves and their patients, medical men are not sufficiently aware of the absorbent powers of the unbroken skin in reference to this poison. One of my pupils, now in practice, informed me of two cases in which medical men applied lotions of corrosive sublimate to their skins. In one instance the corrosive sublimate was used in the proportion of eight grains to one ounce of spirit. It produced immediate and intense irritation, followed by vesication and suppuration of the skin. He suffered for several days from irritative fever and tenesmus. In the second case, from one to two fluid drachms only of a similar solution were used, and the parts were immediately bathed. In spite of this, similar local and constitutional symptoms followed. If applied to an abraded or ulcerated skin, corrosive sublimate would produce all the effects of acute poisoning.

Mr. Annan has reported a case in which the local action of corrosive sublimate appears to have led to death after a long period. In Jan. 1845, a shepherd, æt. 38, had been employed several hours daily in washing sheep affected with cutaneous disease, with a solution of two drachms of corrosive sublimate in twenty ounces of water, in which muriate of ammonia was also dissolved. He was suddenly seized with sickness, vomiting, constitutional irritation, and after the lapse of five days, with salivation, although not severe. He did not recover for a fortnight. In six weeks he experienced a similar attack from the same cause, and this left behind it great debility and emaciation. He resumed his occupation, but was attacked with wandering pains in the joints and diseases of the bones,—as if from the secondary effects of mercury; and he died fourteen months after the first attack. (Med. Times, July 25, 1846, p. 331.) Of ten of the sheep, two died shortly after the application. Salivation is a common effect of the external application of this poison. Dr. Guerard has seen ptyalism produced as a result of three corrosive sublimate baths (one ounce of the poison to about ten gallons of water), taken at intervals of three days; but the effects produced by the solution are never so powerful or so dangerous as those which arise from the application of the poison in the form of ointment. There are many ointments sold by quacks, for the treatment of skin diseases, which contain corrosive sublimate.

When any mercurial preparations are used as caustics, salivation may very speedily follow. Breschet observed this effect in twenty-four hours from the application of the acid nitrate of mercury to the cervix uteri.

APPEARANCES AFTER DEATH.—These, as in the case of arsenic, are chiefly confined to the stomach and bowels; but the mouth, throat, and gullet, are commonly more or less visibly affected. The mucous membrane is softened, of a white or bluish-gray color, and sometimes inflamed; in advanced cases, it is found peeling off: that which lines the œsophagus is similarly affected, and partially corroded and softened. The mucous membrane of the stomach is more or less inflamed, sometimes in patches; and there are masses of black extravasated blood found beneath it. Occasionally the whole cavity is stated to have presented a a slate-gray color from the partial decomposition of the poison by the membrane itself; and beneath this the mucous coat may be found reddened. This gray tint of the mucous membrane has been considered by some to be a special indication of the action of the poison on the living mucous membrane; but it is not always present. A case occurred at Guy's Hospital, in which the mucous membrane was simply inflamed, and resembled the condition presented in cases of arsenical poisoning. In a case which proved fatal on the fifteenth day, the mucous membrane had a dull slaty appearance (ante, p. 402). In another case the inner coat presented a deep yellow tint (from bile), with only slight redness of the folds. M. Lassaigne describes a fatal case in which the stomach had a deep violet-red color, and there was an effusion of blood in the course of the vessels; but there was no ulceration. The slate-gray tint described by Orfila and delineated by Roupell, from their experiments on animals, was not observed; and it is a matter for consideration whether this may not be one of those appearances which are liable to mislead those who rely so exclusively on the results of experiments on animals. (See Ann. d'Hyg. Juillet, 1858, p. 204.) The coats of the stomach are sometimes corroded, and so much softened, that they cannot be removed from the body without laceration. Similar appearances have been met with in the intestines, especially in the cæcum. In Dr. Herapath's case, in which a scruple was taken, and death occurred on the ninth day, the mucous membrane of the stomach was softened; but there were no well-marked appearances of the chemical action of the poison in this organ. The cæcum had been the seat of the most violent inflammation, the whole surface being of a deep black-red color, and there were patches of sloughing in the coats. (Lancet, Dec. 27, 1845, p. 700.) In a case which occurred to Dr. Thomson, of Perth, in which a man died forty hours after having swallowed two drachms of corrosive sublimate in powder, the mucous membrane of the stomach, duodenum, upper portion of the ileum, and parts of the large intestines, were found of a bright red color. This appearance was most marked at the cæcum and sigmoid flexure of the colon. The local action of the poison on the mouth and fauces was in this instance considerable. There was no suppression of urine. (Edinburgh Monthly Journal, Dec. 1851, p. 532.) Perforation of the stomach is rare as an effect of this poison. There is, I believe, only one case recorded in which the appearance was found. Certain morbid changes have been met with in the urinary and circulating organs; and Mr. Swan states that he has found the ganglia and branches of the sympathetic nerve inflamed; but these changes are not by any means characteristic of this variety of poisoning. Appearances in the alimentary canal, like those just described, have been seen not only where the case has terminated fatally in a few hours, but where it has been protracted for six, eight, and even eleven days. (Chaussier, Recueil de Mémoires, 363.) In chronic cases, inflammation of the salivary organs, with ulceration of the gums, is met with.

In the case of J. W. (ante, p. 400), on examining the body twenty-two hours after death, there were the following appearances. At the greater curvature of the stomach, within four inches of the intestinal opening, there was a large patch of inflammation, about the size of the palm of the hand. The mucous membrane was filled with red blood, and presented marks of inflammation throughout. The slate-gray color, which has been seen in some cases of poisoning by corrosive sublimate, did not here exist. There was no appearance of corrosion or ulceration in any part. The small intestines were healthy, with the exception of slight inflammation of the mucous membrane about the lower two-thirds, and this was more marked towards the termination of the intestine. Near to the cæcum there were several patches of inflammation. The whole of the large intestines were highly inflamed; and there were several small spots of ulceration in them, about the size of a pea. The liver and spleen were congested, the gall-bladder contracted and containing scarcely any trace of bile. The bladder was much contracted; the mucous membrane slightly injected: it contained about half an ounce of turbid urine. The mucous membrane of the gullet was reddened from inflammation, but had no other abnormal appearance. The lungs were œdematous, somewhat firm and doughy; at the base of the right lung there was inflammatory œdema. The bronchial membrane was inflamed throughout its whole extent; and within the tubes there was an abundance of frothy mucus. The heart was rather smaller than natural; but the cavities and the muscular substance were healthy. Some partially coagulated blood was found in them.

In the case which proved fatal on the fourth day (ante, p. 401), the body was inspected sixteen hours after death. The membranes of the brain were opaque, there was a large quantity of fluid beneath the arachnoid. The brain was remarkably pale. The lungs were somewhat consolidated, as if from early pneumonia. The heart was healthy, and its cavities were filled with colorless firm fibrin. The kidneys were congested. The bladder was empty and contracted, and there were some small pink spots on its mucous membrane. The mucous membrane of the gullet had a vermilion hue. The stomach presented a pink color on its inferior surface, near its middle. The small intestines were healthy, and lined with a thick yellow mucus. The cæcum and ileo-cæcal valve showed signs of the most intense inflammation; some portions were of a deep purplish-black color, with patches of sloughing mucous membrane, tinged green by fæces. The colon and rectum also exhibited traces of the most violent inflammation, especially the ascending and transverse portions. Here were found oval patches of sloughing mucous membrane, about the size of small almonds, and tinged green by fæces passing over them. The condition of the cæcum here described was found in Dr. Herapath's case (ante, p. 399).

In Mr. Wade's case (ante, p. 402), the lining membrane of the mouth and gullet was quite healthy. The mucous membrane of the stomach, to the extent of three inches from the cardiac opening, was converted into a gangrenous mass, having a corroded, ragged appearance, of a dusky-brown color, approaching to black. Around this the mucous coat was reddened; but it was healthy towards the intestinal opening. There were no morbid changes of any note in the intestines. The cavities of the heart were empty. The whole of the mucous membrane of the air-passages was in a state of extreme congestion, varying from a deep-red to a purple colour; the smaller air-tubes being filled with a frothy, bloody fluid. As there had been no sign of cerebral disturbance in this case, the head was not examined. (Med. Gaz. vol. xli, 1848, p. 780.)

Fatal dose.—It is difficult to state this with any degree of certainty, since it is only by accident that the quantity taken can be ascertained, and the fatal effects must vary according to many circumstances. A child, aged 3 years, died in twenty-three days from the effects of twelve grains of corrosive sublimate. The *smallest* dose which is reported to have destroyed life was *three* grains. This was also in the case of a child, and the quantity was accurately determined from the fact of its having been made up by mistake for three grains of calomel, which the physician intended to order. (This case is referred to in the Lancet, 1845, p. 297.) A very loose and imperfect report, either of the same or of a similar case, is given in the Ann. d'Hyg. 1835, i, 225. It is stated that three children lost their lives. In the case of Reg. v. Robertshaw (Carlisle Lent Assizes, 1845), there is reason to believe that two, or not more than three grains, were taken, and proved fatal to an adult. (Med. Gaz. xxxv, 778.) In its power as a poison, it is therefore somewhat similar to arsenic. Persons have been known to recover who have taken very large doses, when remedies were timely administered, or when there was early vomiting. I have elsewhere reported a case of recovery from a dose of nineteen grains in a girl, æt. 18. (Guy's Hosp. Rep. 1850, p. 213.) In an instance reported in the Journal de Pharmacie, a man recovered in three days after having taken one drachm of the poison; and a case of recovery from a similar dose is described in the Edinburgh Monthly Journal, 1850 (p. 380). In the Medical Gazette (xiv, 63), Dr. Booth mentions a case in which *an ounce* of corrosive sublimate had been swallowed after a full meal; and by timely vomiting the subject of this rash act escaped with comparative impunity. In a case by Dr. Percy (Med. Gaz. xxxi, 942), a girl, aged 17, mixed thirty grains in coarse powder with water in a teacup, and then swallowed the liquid. A considerable quantity remained in the cup. Symptoms of poisoning came on, but the girl recovered. Dr. Percy doubted whether any of the poison had reached the stomach.

A case of recovery, after *forty grains* had been taken in whiskey, under circumstances favorable to its fatal operation—*i. e.* on an empty stomach—is recorded by Dr. Andrews. (Cormack's Journal, Feb. 1845, 102.) The patient was a woman, æt. 65. The actually smallest dose required to destroy an adult, under ordinary circumstances, cannot therefore be determined at present from any reported facts. The medicinal solution is used in doses varying from one thirty-second part to one-eighth of a grain. Hence, in potency, corrosive sublimate may be considered as not inferior to arsenic; and a fatal dose, under circumstances favorable to its operation, may be assumed to be about *three grains.* This quantity has actually destroyed the life of a child. Its effects upon adults has not yet been determined as a matter of experience; but, independently of the fatal case above referred to, in which this dose was supposed to have been taken, there is one quoted by Dr. Beck, in which six or eight grains destroyed the life of an adult. (Med. Jur. vol. ii, p. 570.)

PERIOD AT WHICH DEATH TAKES PLACE.—In an acute case of poisoning an individual commonly dies in from one to five days. But death may take place much sooner or much later than this. A person has been known to die from the effects of this poison in eleven hours (Christison, 402); and in one instance, of a child two years old, by whom twelve grains had been taken, death probably occurred in six hours. (Niemann's Taschenbuch, 451.) A case is reported in which a child, aged 7, was killed in three hours by eighteen grains of corrosive sublimate. In the following instance reported by Mr. Illingworth, the period, although inferential, was probably even shorter. A man, æt. 30, was found dead on the 4th of December, 1842, at half-past seven A.M. He had vomited some half-digested food, mixed with blood and mucus. On a shelf near him was a drinking-horn, containing about three drachms of corrosive sublimate. It was ascertained at the inquest that he had died from the effects of this poison. He had put water into the drinking-vessel, and had probably swallowed the poison while thus loosely suspended;—the exact quantity taken could not be ascertained. The deceased was last seen alive at half-past eleven the preceding evening, therefore only eight hours before he was found dead. When discovered, the face and the extremities were cold. From all the circumstances it was inferred that, even admitting the deceased to have taken the poison immediately after he was last seen alive, he could not have been dead less than six hours. This would carry the duration of the case to *two hours* from the time of taking the poison. (Med. Gaz. xxxi, 557.)

The most rapidly fatal instance of poisoning by corrosive sublimate hitherto recorded, was communicated to me by Mr. Welch. In June, 1846, a man mixed some corrosive sublimate (quantity unknown) with some tea, and drank it. The symptoms which followed were a sensation of burning heat in the mouth and mucous vomiting. He was insensible when seen; and from the circumstances of the case, he must have died in less than *half an hour*.

On the other hand, a case may be protracted for several days. The following summary will not only show this, but will also prove that the time at which the poison destroys life cannot be inferred from the quantity taken. In an instance referred to by Niemann (Tasch. d. Arzniew. 452), one ounce of the poison was swallowed, and the person did not die until the sixth day. In a case related by Dr. Venables, two drachms of the poison killed a woman in eight days. In one reported by Sobernheim, three drachms did not kill for eleven days. A case is quoted by Beck (Med. Jur. ii, p. 570), in which a man who had taken only six or eight grains in solution, survived until the twelfth day. The longest case of acute poisoning which I have met with is that which is elsewhere referred to (ante, p. 402), in which death did not take place until the *fifteenth day*. In death from chronic poisoning, the case may be protracted almost indefinitely.

TREATMENT.—If vomiting does not already exist, it must be excited by the use of emetics. (See Treatment of Arsenic, ante, p. 344). Various chemical antidotes have been suggested for this poison; and among these, albumen, both of the yolk and white of egg, mixed with water, and administered in large quantity, is perhaps the best fitted to counteract its effects. This remedy appears to have been beneficial even when it was not taken until some time after the poison had been swallowed; but too much reliance must not be placed on it. The removal of the poison from the body should be the great object of our treatment. Gluten may also be used. This may be prepared by washing flour in a muslin bag under a current of water. Should the case be urgent, the flour may be at once exhibited in the form of a thick paste mixed with milk or water. Gluten may be obtained when albumen is not at hand. M. Bouchardat states that Cullerier saved two hundred patients who had taken an over-dose of corrosive sublimate, by making them swallow, in twenty-four hours, from seven to eight quarts of milk, with a decoction of linseed and warm water! (Gaz. Méd. Jan. 9, 1847.) These antidotal liquids may be serviceably employed for the purpose of favoring the expulsion of the poison by vomiting, on which the safety of the patient essentially depends.

Among the numerous chemical antidotes proposed, M. Mialhe has recommended the hydrated protosulphuret of iron; but Orfila has proved that this substance is totally inefficacious when not given until after the lapse of ten or fifteen minutes from the time at which the poison has been swallowed. (Toxicologie, i, 720.) The same objection holds to the hydrated persulphuret recommended by M. Bouchardat.

In all cases, the entire expulsion of the poison from the stomach should be looked to by the practitioner; and albumen or gluten may be given at the same time to aid the efforts of vomiting. The use of the stomach-pump is of questionable propriety; since, if the gullet and stomach are much softened and corroded, very slight force in its employment might lead to perforation. In order to check excessive salivation, as a subsequent symptom, Mr. Allison has recommended small doses of chlorate of potash. (See Med. Gaz. xxxviii, 953.)

CHAPTER XXVII.

CHEMICAL ANALYSIS IN THE SOLID STATE—REDUCTION-PROCESS—TESTS FOR CORROSIVE SUBLIMATE IN SOLUTION—OBJECTIONS TO THEIR EMPLOYMENT—IN ORGANIC LIQUIDS AND SOLIDS—DETECTION OF MERCURY IN THE TISSUES—ITS ENTIRE DISAPPEARANCE FROM THE BODY—QUANTITATIVE ANALYSIS—CALOMEL—WHITE AND RED PRECIPITATES—VERMILION—CYANIDE OF MERCURY—TURBITH MINERAL—NITRATES OF MERCURY.

CHEMICAL ANALYSIS.

In the solid state.—We will first suppose that the poison is in the *solid* state, and in the form of a white powder. 1. A small quantity heated on thin platina foil or mica, is entirely volatilized at a moderate heat—(care should be taken in performing this experiment),—in this property, corrosive sublimate resembles arsenic, but differs in all other respects. 2. When the powder is heated in a small reduction-tube, it melts (unlike arsenic), and boils at about 560°, and its vapor is condensed unchanged in stellated groups of prismatic crystals, on the cold part of the tube. These crystals are beautifully defined under a low power of the microscope. 3. It is very soluble in water (ante, p. 398), a portion floating in the cold, but if the water be warmed, the powder will be speedily dissolved. 4. The powder is readily dissolved by ether, and the solution, spontaneously evaporated in a watch-glass, leaves white prismatic crystals. Ether, shaken with the watery solution, removes the greater part of the corrosive sublimate. 5. It is dissolved by hydrochloric acid, strong or diluted, and a copper-wire plunged into the solution *in the cold*, is covered with a white silvery deposit. 6. A small quantity of the powder dropped into a white saucer, containing a solution of the iodide of potassium, is turned of a bright scarlet color. This may be applied to the sublimate obtained under 2, or to the crystals under 4. 7. Dropped into potash in a similar way, it is turned of a yellow color. It is insoluble in and decomposed by potash. 8. A solution of hydrosulphuret of ammonia, or of sulphuretted hydrogen, produces at first a yellowish and afterwards a black compound. 9. When a few grains are rubbed on a clean surface of copper, with a mixture of one part of muriatic acid, and two parts of water, a bright silvery stain of mercury is produced, which is entirely volatilized by heat. If pure zinc or pure tin-foil be used instead of copper, the surface acquires a silvery lustre, and the metal is rendered remarkably brittle. 10. When mixed with three or four parts of calcined (anhydrous) carbonate of soda, and heated in a small tube similar to that employed in the analysis of arsenic, the metal is reduced; and a ring of bright globules of mercury is formed, while common salt remains in the tube. For the success of this experiment, the materials must be quite dry, and the tube at first gently heated; any undecomposed corrosive sublimate that may be sublimed, should be driven higher up, or even out of the tube, before finally applying a strong heat, so that the ring of mercury may not be obscured by it. The last experiment is conclusive of the nature of the substance; because *mercury*, being the only liquid metal, is the only metal which sublimes in *globules*. If the globules are not visible to the eye, they may be plainly seen by the aid of a microscope. They are identified by their bright silvery lustre under reflected light, their complete opacity by transmitted light—and by their perfect sphericity. A globule of the 16,000th of an inch in diameter may be thus recognized. It is impossible to confound this sublimate, in isolated bright opaque globules, with that of the angular transparent crystals of arsenious acid (ante, p. 347); nevertheless, when any doubt exists, the mercurial nature of the sublimate may be thus proved. File off the ring of glass containing the sublimate. Heat it in a short wide tube with a few drops of nitric and muriatic acids mixed. Evaporate to dryness on a sand-bath, at the lowest temperature. A white residue, or a sublimate (corrosive sublimate), will be obtained in the tube, according to the heat applied. This, when touched with a strong solution of iodide of potassium, produces a rich scarlet color. (Exp. 6, supra.)

Another mode of identification consists in dropping into the tube containing the sublimate, a crystal of pure iodine, and closing the mouth of the tube. At a temperature of about 60° to 70°, a chemical union takes place, and a ring of red iodide of mercury is slowly formed in the tube, occupying the place of the metallic globules. If the end of the reduction-tube, containing the fused chloride of sodium left as a residue of the decomposition, be filed off, reduced to powder, and boiled in water with a little diluted nitric acid, a solution is obtained in which, on the addition of nitrate of silver, *chlorine* may be proved to exist. The analysis is then complete. The properties mentioned under 1, 2, and 5, are possessed in common by other bodies; but the other characters are peculiar to the persalts of mercury: and when the results agree, they render it absolutely certain that the powder must be a persalt of that metal. The action of nitrate of silver upon the solution of the residue will prove that the persalt must have been a *chloride*, and the only soluble chloride is corrosive sublimate. There are, therefore, no *objections* to this mode of analysis. The experiment of reduction will answer with any visible quantity of the poison. A good reducing agent was proposed by the late Dr. Frampton, namely, pulverulent metallic silver; but this is not so easily procurable as dry carbonate of soda. Four parts of this may be used to one of corrosive sublimate. (See Med. Gaz. xxxii, 384.)

The *weight* of a mercurial sublimate may be determined by the same method as that of arsenic (p. 349, ante), and it may be preserved in like manner, *i. e.* by hermetically sealing the tube.

In solution in water. Liquid tests.—Corrosive sublimate is very soluble in water (ante, p. 398), forming a clear solution, which, when concentrated, has a faintly acid reaction, and a strong metallic taste. A few drops of the solution may be gently evaporated on a slip of glass, and then set aside to crystallize. If it be corrosive sublimate, it forms slender opaque silky prisms intersecting each other, and sometimes of considerable length. When touched with a solution of iodide of potassium, these crystals acquire a bright scarlet color (ante, p. 414). These characters, which may be obtained from the minutest crystal and only one drop of solution, prove that the body dissolved in water is corrosive sublimate; it is thus distinguished from every other mineral poison, and all other substances whatever. 1. *Potash.*—On adding a small quantity of a solution of potash to the solution, a reddish-colored precipitate falls, becoming yellow by the addition of a larger quantity of alkali. This precipitate, when washed, dried, and heated in a reduction-tube, yields a well-defined ring of metallic mercury. The filtered liquid, on being tested with nitrate of silver, will be found to contain *chloride* of potassium, thus proving that the mercury was combined with chlorine, and that the compound was soluble in water. 2. *Chloride of tin.*—On adding this test in rather large quantity to the solution, a white precipitate at first falls down (calomel), becoming speedily of a slate-gray color, and afterwards almost black. On warming the liquid, it soon becomes clear, while a heavy precipitate, in great part formed of pure metallic mercury, falls to the bottom of the vessel. The mercury may be collected by pouring the liquid on a filter, and afterwards warming the filter; or its presence may be easily demonstrated in a test-tube, by draining the water carefully from the precipitate, and then forcing down upon this a slip of bibulous paper: this absorbs the water from the mercury, and the pressure condenses the metal into one or more well-defined globules. 3. *Sulphuretted hydrogen gas.*—This produces at first a precipitate, partly black and partly white (chloro-sulphuret), becoming entirely black when the current of gas has been allowed to pass in for some time. *Hydrosulphuret of ammonia* produces a similar precipitate in the solution,—thus clearly distinguishing corrosive sublimate from arsenic. The test acts equally in an acid solution of the salt. The precipitated black sulphuret of mercury, dried and heated with carbonate of soda or metallic silver, furnishes globules of pure metallic mercury, and is thus known from other black sulphurets. Nitrate of silver, added to the diluted solution of corrosive sublimate, causes a white precipitate of *chloride* of silver, insoluble in nitric acid. This demonstrates the chlorine, while Exp. 2, demonstrates the mercury. These tests are alone sufficient to show that the substance dissolved is corrosive sublimate.

The chloride of tin, with the examination of the deposit on the filter by the microscope, may be regarded as the most delicate of the liquid tests for mercury, but the liquid tests for the detection and separation of this metal are less delicate in their reaction than some of the metals, either used singly or in combination. Copper, tin, zinc, and silver in foil or a finely pulverulent state, easily remove mercury from a solution of corrosive sublimate when in small quantity, especially if the solution be slightly acidulated and boiled. Of these metals copper is to be preferred, because by reason of its red color a very small amount of deposit is easily distinguished upon it. If we acidulate the suspected solution of corrosive sublimate with a few drops of diluted hydrochloric acid, and introduce a slip of bright copper, or what is better, fine copper-gauze (ante, p. 414), it is soon coated with metallic mercury, having more or less of a silvery lustre, especially on friction. On heating the copper, previously well washed in alcohol, in a reduction-tube, the mercury may be obtained in well-defined globules, and these can be examined by a lens or the microscope. If the corrosive sublimate is present only in very minute proportion, the liquid must be boiled before a deposit will take place.

Reinsch found that one part of corrosive sublimate in 1000 parts of water, mixed with hydrochloric acid, gave immediately, to a surface of copper, a white metallic film: in 5000 parts of water, without the addition of hydrochloric acid, there was no deposit in the cold, but the copper acquired a golden-yellow color on boiling. When boiled in a solution to which hydrochloric acid has been added, it turned gray, becoming covered with a thin film of mercury even when the quantity of water amounted to from 12,000 to 15,000 parts. (Gmelin's Chemistry, vol. vi, p. 57.) Although these experiments refer rather to the effect of dilution than the smallness of quantity detected, I entertain no doubt from the results which I have obtained, that copper thus employed constitutes the most delicate test for mercury in corrosive sublimate. Neither this nor any other metal enables us to detect the chlorine.

Gold foil or wire immersed in the acidulated solution produces no change whatever; but if a slip of zinc foil, or a piece of zinc wire is merely brought in contact with it, or a zinc wire is twisted round the gold and immersed in the liquid feebly acidulated with hydrochloric acid, there is sooner or later, by galvanic action, a deposit of mercury on both metals: the gold acquiring a grayish-white appearance. The metal with the deposit should be washed in alcohol or ether, dried and heated in a tube, when mercurial globules will be obtained. By either of these processes I have found, that a quantity of corrosive sublimate amounting to about the 144th part of a grain diffused in 8000 times its weight of water, would, in twenty-four hours, give evidence of the presence of mercury. This appears to me to be near the limits of the process for detecting mercury.

Objections.—Among the above-mentioned tests, there is only one to which any objection can be offered, namely,—3. Sulphuretted hydrogen gas, or hydrosulphuret of ammonia. Either one or both of these reagents will give a black or dark brown precipitate with several metals, as for example with the salts of lead, copper, bismuth, silver, nickel, iron, and tin. The black precipitate derived from corrosive sublimate is, however, distinguished from all the others, by the fact that it easily yields mercury when dried and heated in a tube with carbonate of soda or metallic silver. But the objection is at once answered by the fact, that the other tests are conclusive of the presence of a *mercurial* compound: it could only apply under the improbable contingency of a black precipitate from sulphuretted hydrogen alone being relied on, as evidence of the presence of corrosive sublimate.

In liquids containing organic matter.—The same process of analysis will apply to the vomited matters and contents of the stomach. Masses of corrosive sublimate may be sometimes locked up in thick viscid mucus and blood; in such cases, the coarse powder being very heavy, it may be sometimes separated by simply agitating the viscid liquid in water, and then decanting it suddenly. This poison, in solution, is decomposed and precipitated by many organic principles, such as albumen, fibrin, mucous membrane, also by gluten, tannic acid, and other vegetable substances. Thus, then, we must not always expect to find it in a state of solution. The liquid should be filtered in order to separate the solid portion; and our object will then be to determine whether any of the poison is held in solution. For this purpose we add to a measured quantity of the liquid, in a long stoppered tube, its volume of ether, and agitate the mixture. On allowing it to stand some time, the greater part of the ether will rise to the surface, holding dissolved a portion of the corrosive sublimate which was contained in the organic liquid.

The ethereal liquid may be poured off, and allowed to evaporate spontaneously in a dial-glass, or other convenient glass vessel. If the poison is present ~~is~~ in large quantity, white prismatic crystals will appear, which are rendered scarlet when touched with a solution of iodide of potassium. The other properties of the solid poison may be brought out by the tests elsewhere described (ante, p. 414). A portion of the crystalline residue may be dissolved in water, filtered through a wet filter, and the appropriate liquid tests then applied. The quantity of corrosive sublimate may be too small for this method of separation; then we may acidulate the liquid with about one-tenth part of its volume of hydrochloric acid, and introduce a slip of copper gauze, at the same time warming the liquid. If the poison be present even in minute quantity, the gauze speedily acquires a silvery-gray color, from a deposit of mercury. It should be well washed in water, in alcohol or ether, again in water, and then dried. On heating it in a reduction-tube, a ring of fine metallic *globules* will appear in a detached form, having a silvery-white lustre. When examined by the microscope, spherical globules are seen, having the properties elsewhere described (ante, p. 415). In order to remove any doubt, the ring of glass on which the sublimate is deposited may be broken up, and the sublimate examined, as already described (ante, p. 415). If the copper test should not give a satisfactory result, the galvanic *gold test* may be applied (ante, p. 417). Dilution interferes with these processes, hence it is desirable to concentrate the liquid as much as possible. One-sixteenth of a grain of corrosive sublimate dissolved in sixteen ounces of water, gave no deposit on gold foil with zinc; but when the quantity was dissolved in one ounce of the organic liquid, metallic mercury was separated and its properties demonstrated in less than half an hour.

Let us suppose that the filtered liquid contains no trace of a mercurial salt, then there is no corrosive sublimate dissolved. We must now direct our attention to the analysis of the *insoluble matters* separated by filtration. These may be boiled in distilled water, the liquid filtered, and tried by agitating it with its volume of ether. It will be found, when the analysis has not been long delayed, that most of the compounds which corrosive sublimate forms with organic matter, yield commonly sufficient poison for detection by boiling them in water. Should water fail to extract the poison, the substance may be brought to dryness and heated with nitro-muriatic acid until all the organic matter is decomposed, and the surplus nitric acid expelled. The residue may then be digested in water, and tested for mercury by the aid of copper-gauze or of gold and zinc. This is also the process to be pursued with organic *solids* supposed to contain the poison. [illegible] discovery of corrosive sublimate in small quantity in an organic liquid (medicine), in the matter vomited, or the contents of a stomach does not necessarily prove that it has been administered as a poison, and with criminal intention. It is sometimes used medicinally; but only in solution, and in very small doses (see ante, p. 412).

Detection of absorbed mercury in the tissues.—The process suggested by Reinsch for the detection of arsenic is equally applicable to the detection of absorbed mercury in the tissues. Although Reinsch neither applied nor proposed it for this purpose, he found that in a clear solution of corrosive sublimate, when the quantity present did not form more than the fifty-thousandth part, globules of mercury might be obtained on copper. (Ann. d'Hyg. 1843, i, 445.) The plan which I have usually adopted is as follows: The liver or other tissue is cut into small pieces, and boiled until the texture is entirely broken up in a mixture of one part by measure of pure hydrochloric acid to six or seven parts by measure of distilled water. A small slip of copper-gauze at the end of a polished copper wire is first introduced. If it acquires a light-gray color, it is probable that mercury is present, and a larger piece of gauze is then introduced. If not immediately coated, the decoction is evaporated with the copper immersed in it. The coated gauze is cleansed, washed, dried (see p. 418), and then heated in a small tube. A minute ring of mercurial globules will be perceptible either to the unassisted eye or by the aid of a lens or microscope. The spherical form, opacity, and metallic lustre by reflected light, are commonly sufficient to identify a mercurial sublimate. If there should be any doubt, the corroborative test recommended in a preceding page may be employed. The copper-gauze, even when it is scarcely changed in appearance by this process, or when it presents only the thinnest film on the red surface of copper, will frequently yield a sublimate of metallic globules when heated. Hence it should be always tested in a reduction-tube before it is rejected.

The metallic deposit, if any, should be examined at once. Mercury is volatile at all temperatures, and in twenty-four hours a thin metallic film may disappear. I have known this to occur with a mercurial deposit in a summer's day;—while arsenic will remain fixed on the metal for many years.

It must be remembered that this result merely proves the presence of *mercury* in the tissues, not of corrosive sublimate. It would show that mercury was present in some tangible form. Whether the substance had acted as a poison or not must be determined from symptoms and appearances;—whether it had been given or intended as a medicine or not would be easily deduced from other circumstances. The only method of proving that the mercury was really in the form of corrosive sublimate, would be either by the discovery of some undissolved portions of the solid poison in the stomach or its contents, or by a separation of the poison itself by ether (ante, p. 418). If dissolved, it would show a soluble salt; all the soluble salts are poisonous, and are rarely used internally as medicines. If undissolved, the absorbed mercury may have been derived from some mercurial medicine innocently taken by the deceased. Nothing is more common than to discover traces of mercury in the liver and tissues, and even in the bile, of dead subjects. No importance is or can be attached to this discovery, except in those cases where there is evidence that a person has actually suffered from symptoms of mercurial poisoning (see ante, p. 67).

Most of the common medicinal preparations of mercury are in an insoluble form. So far as the tissues are concerned, the chemical result would be the same whether the mercury was taken in a dissolved or undissolved state. In the case of Reg. v. Bacon (Lincoln Summer Assizes, 1857), sublimates of arsenic and mercury were obtained by this process from the viscera of deceased, the spherules of mercury being visibly intermixed with the octahedral crystals of arsenious acid, as seen by the microscope. Arsenic had been criminally administered to the deceased; but with respect to the mercury, it was ascertained that the deceased had taken medicinally two grains of calomel two days before her death. The body had been buried twenty-one months, and, as the analysis proved, some mercury was still remaining in the viscera after this long period. This is sufficient to show that the process by copper is both delicate and certain.

A person may die from the effects of corrosive sublimate, and no mercury may be found in the tissues. A case of this kind occurred to me some years since at Guy's Hospital; and another, in which the deceased died in fifteen days from a large dose of corrosive sublimate in whiskey, has been reported by Dr. Geoghegan. On this occasion, although the local effect of the poison on the throat, stomach, and bowels, were of an intense kind, the viscera, on careful analysis, yielded no trace of mercury; it had been entirely eliminated (see ante, p. 402; see also Med. Gaz. vol. xlvi, p. 253).

Corrosive sublimate is not easily lost from organic liquids, when it is in moderate quantity. A few grains of this poison were mixed with some ounces of albumen, gruel, and porter, in January, 1839. An abundant precipitate was formed. The mixture was exposed for eight years; but the poison was, at the end of that time, easily discovered by the galvanic gold test, both in the supernatant liquid and in the precipitate. Corrosive sublimate is not always found in the stomachs of persons poisoned by it, although, from its readily combining with the mucous membrane, it is more likely to be detected than arsenic. In a well-marked case, which occurred to Mr. Watson, in which two drachms killed a person in six days, none was found on a chemical analysis of the contents. In a case in which two drachms were swallowed, and the man died in four days, no mercury was detected in the stomach or tissues (ante, p. 401). In the case of J. W., elsewhere referred to (ante, p. 410; also p. 67), in which a similar quantity was taken, and death occurred on the *fourth* day, the stomach and tissues were examined by the galvanic gold test; but not a trace of mercury could be detected in them. In another, which occurred to Dr. Wegeler, in which a young man poisoned himself with three drachms of corrosive sublimate, and died on the *sixth* day, none of the poison could be detected in the stomach and intestines. (Canstatt's Jahresherichṭ, für 1846, Bd. v, p. 81; and Wharton and Stille's Med. Jur. 1855, p. 440.) In Dr. Geoghegan's case, in which a large dose was taken, and the man died on the *fifteenth day*, the mouth, stomach, and intestines, as well as liver, spleen, and kidneys, were submitted to analysis; but there was no trace of mercury, either in the free or absorbed state. (Reg. v. Walsh, Med. Gaz. 1850, vol. xlvi, p. 255.) Referring to what has elsewhere been said (ante, p. 68) on the elimination of mercury, these facts corroborate the view of M. L. Orfila—namely, that in acute poisoning by this mineral, if a person survives *fifteen days*, it is probable that no trace of mercury will be found in the body. "The experts will, however," he remarks, "commit a gross error if they conclude from this that there has been no poisoning." (Comptes Rendus, Jan. 15, 1852; and Wharton and Stille, Med. Jur. p. 441.)

The diffusion of this metal through the system, even when mercurial preparations have been applied externally, is well illustrated by the fact that Landerer detected mercury in the pus from the bubo of a man who had employed mercurial frictions. (Heller's Archiv, 1847, H. 2, p. 185.)

QUANTITATIVE ANALYSIS.—If the poison be entirely in a soluble form, we may procure the mercury from a part only by the use of ether, and calculate the remainder proportionably. If it be in an insoluble form, we must then pursue the process elsewhere described, and precipitate it entirely by chloride of tin, purifying the mercury by boiling it first in potash, and secondly in hydrochloric acid. For every 100 grains of metallic mercury obtained, we must allow 135 grains of crystallized corrosive sublimate to have been present. Lassaigne advises the use of sulphuretted hydrogen. (Ann. d'Hyg. Juillet, 1858, p. 204.)

CALOMEL.

This substance also called chloride, or subchloride of mercury, although commonly regarded as a mild medicine, is capable of destroying life, even in comparatively small doses. Several cases have been already referred to, in which excessive salivation, gangrene of the salivary organs, and death, have followed from the medicinal dose of a few grains (p. 408). There is a case reported in the Medical Gazette (xviii, 484), in which a boy æt. 14, was killed in about three weeks by a dose of only *six grains* of calomel. It is singular that in this case neither the teeth nor the salivary glands were affected; still, considering the effects of calomel in other instances, it seems most probable that the ulceration and gangrene of the face which followed were due to it. Pereira mentions the case of a lady who was killed by a dose of twenty grains of calomel. She had previously taken a moderate dose without a sufficient effect being produced. Sobernheim states that a girl, æt. 11, took, in twenty-four hours, eight grains of calomel for an attack of croup, and died in eight days from inflammation and ulceration of the mouth and throat. In another instance, which occurred to Lesser, fifteen grains of calomel produced similar effects, with excessive salivation; and this patient also died in eight days. Meckel relates that twelve grains have destroyed life. (Lehrbuch der Ger. Med. 267.) Two cases of death from calomel, in children, are recorded in the "Registration Returns for 1840."

There are many other fatal cases on record, and the facts seem to leave no doubt that calomel may, in large doses, act as an irritant poison. It was supposed that these effects might be ascribed to this compound being adulterated with corrosive sublimate; but this supposition is not well founded. It has also been suggested that calomel might be converted to corrosive sublimate by the free hydrochloric acid present in the stomach; but the small proportion in which this acid exists is adverse to this suggestion. (Ed. Med. and Surg. Journ. vol. xlix, p. 336.)

Analysis.—1. Calomel is insoluble in water, alcohol, and ether. By either of the latter liquids corrosive sublimate may be detected in and separated from it. 2. When heated on platina or mica, it is entirely volatilized. 3. Potash and ammonia render it black. 4. Heated with dry carbonate of soda, it yields a sublimate of metallic mercury in globules and leaves a white residue of chloride of sodium, which may be examined by the same process as the residue of corrosive sublimate (ante, p. 415). Owing to its great weight and insolubility, calomel may be separated from *organic liquids* and solids by simply washing them and decanting the water.

WHITE PRECIPITATE. AMMONIO-CHLORIDE OF MERCURY.

Symptoms and effects.—This is an irritant compound, which is much used by the poor as a local application for ringworm. In January, 1840, a young woman who had swallowed this substance was received into St. Thomas's Hospital. She had mixed it and taken it in water,—but the quantity swallowed could not be ascertained. The stomach-pump was employed, mucilaginous drinks and olive-oil were administered; and in the course of a few days she perfectly recovered. The symptoms under which she suffered were those of irritation of the stomach. One instance of death from salivation produced by this compound is recorded in the Registration Returns for 1840, in a child, aged seven; and within the last two years there have been several attempts at murder by the administration of white precipitate. Mr. Procter, of York, has communicated to me a case in which a woman recovered after having taken forty grains. In half an hour she complained of pain in the gullet extending to the stomach, and her mouth was dry and clammy: the inside of the lips and tongue was slightly blistered. There was neither vomiting nor purging until a dose of tartar emetic and castor oil had been given. In about three days, she recovered. A case occurred to Mr. Michael, of

Swansea, in which a woman, æt. 37, swallowed not less than *one hundred grains* of white precipitate. She was seen in three hours and a half: she complained of great pain in the stomach, with cramps of the left side and lower limbs, coming on at intervals of two or three minutes. The pulse was rapid, weak and thready, the surface cold and clammy, and the tongue red. She had vomited a thick white tenacious mucus with a white sediment: vomiting was kept up for two hours. The bowels were freely opened. There was great prostration, continuing for several hours, and the pain in the stomach remained for three or four days, after which it gradually subsided (Brit. Med. Journal, Oct. 31, 1857, p. 909). In these two cases there was no salivation, but in one which occurred in the practice of Mr. Giles, this was a prominent symptom. A girl swallowed about half a drachm of white precipitate in a cup of tea. She suffered much pain in the stomach, and there was frequent purging. On the following day there was swelling of the face and gums with salivation, which lasted several days. She recovered in about nine days (Lancet, 1857, vol. ii, p. 9). Judging from these cases, white precipitate cannot be regarded as an active poison; and its effects are somewhat uncertain. It must, however, be ranked among mercurial poisons. In a case tried at the Assizes a few years since, a woman owed her acquittal to the assumption that this was not a poison. There have, however, been several convictions since. At the Exeter Lent Assizes, 1855, a boy was convicted of the attempt to administer this poison to his father (Reg. v. Daniel).

Analysis.—This powder resembles corrosive sublimate in being entirely volatilized by a moderate heat, and in giving a metallic sublimate with dried carbonate of soda, but it differs from it and resembles calomel in being insoluble in water. It differs from calomel in remaining white when mixed with solution of ammonia. By heating it with a solution of potash, ammonia is evolved, chloride of potassium is produced, and yellow oxide of mercury, after long boiling, is left. It is soluble in strong nitric acid, and chlorine may be detected in the solution by nitrate of silver. The chloride of tin or the process by copper will separate the mercury (see ante, p. 419).

RED PRECIPITATE. RED OXIDE OF MERCURY.

Symptoms and effects.—This substance is poisonous, but instances of poisoning by it are very rare. The following case occurred at Guy's Hospital in 1833. A woman, æt. 22, who had swallowed a quantity of red precipitate, was brought in laboring under the following symptoms: The surface was cold and clammy, there was stupor approaching to narcotism—frothy discharge from the mouth, and occasional vomiting: the vomited matters contained some red powder, which was proved to be red precipitate. There was considerable pain in the abdomen, increased by pressure; and there were cramps in the lower limbs. On the following day the mouth and throat were painful, and the woman complained of a coppery taste. The treatment consisted in the use of the stomach-pump and the free administration of albumen with gluten. She left the hospital in four days, still under the influence of mercury. The quantity of oxide here taken was not ascertained. Mr. Alison met with a case in which thirty-five grains were taken. Emetics were given, and the stomach-pump was used. The woman gradually recovered, having suffered from a burning pain in the stomach. (Lancet, N. S. vol. xix, p. 401.) Sobernheim relates a case in which a man, æt. 26, swallowed an ounce of red precipitate. He was speedily attacked with pain in the abdomen, nausea, purging, cramps, and general weakness. The vomited matters consisted of masses of mucus containing red precipitate. He continued to get worse, and died in less than forty-eight hours after taking the poison. On inspection, the mucous membrane was found eroded and inflamed in patches,—small particles of the poison being imbedded in it. The duodenum was in a similar state, and there was a large quantity of red precipitate in the contents of this intestine, as well as in the stomach. (Op. cit. 250.)

A common opinion exists among the vulgar, that this compound is possessed of very active poisonous properties; hence it is sometimes administered with criminal design.

Analysis.—Red precipitate is known,—1. By its being in red crystalline scales. 2. By its insolubility in water,—this, together with its great weight, renders it easy of separation from *organic liquids*. 3. It is readily dissolved by warm muriatic acid, forming a solution possessing all the properties of a solution of corrosive sublimate (see ante, p. 416). 4. When heated in a small tube, it becomes black (reacquiring its red color on cooling); and, while an abundant sublimate of mercury is formed, oxygen gas is evolved.

CINNABAR. VERMILION. PERSULPHURET OF MERCURY.

The term *Cinnabar* is applied to a dark and heavy compound of sulphur and mercury, while *Vermilion* is the same substance reduced to a fine powder. It is well known as a red pigment, and is often employed in coloring confectionery and wafers. I have not been able to find any instance of its having acted as a poison on man. Orfila believes that it is not poisonous. It has, however, proved fatal to animals in the proportion of from thirty to seventy grains, even when applied externally to a wound. Cinnabar is sometimes used for giving a red color to ointments, *e. g.* the sulphur ointment. In such cases the quantity is very small, and can do no injury even if swallowed.

Dr. Sutro has published a short abstract of a case in which the *vapor of vermilion* applied externally produced severe symptoms. A woman, by the advice of a quack, applied this vapor to a cancerous breast. She employed three drachms of vermilion, covering herself with a sheet, so that the vapor should only reach the body externally. After three fumigations, she suffered from severe salivation and violent fever, which continued for four weeks. The right arm became œdematous. (Med. Times, Sept. 27, 1845, p. 17.)

CYANIDE OF MERCURY.

Symptoms and effects.—This is a substance which is but little known except to chemists, yet it is an active poison, and has caused death in at least two instances. In April, 1823, a person who had swallowed *twenty grains* of this compound (thirteen decigrammes), was immediately seized with all the symptoms of poisoning by corrosive sublimate, and died in nine days. There was continued vomiting, with excessive salivation, ulceration of the mouth and fauces, suppression of urine, purging, and lastly, convulsions of the limbs. On inspection, the mucous membrane of the stomach and intestinal canal was extensively inflamed. (Orfila, i, 583.) Dr. Christison quotes a case in which *ten grains* destroyed life within the same period of time. (On Poisons, p. 427); and in this case the symptoms were severe irritation of the stomach, inflammation of the mouth, and suppression of urine. These facts are adverse to the theory of Bernard, namely, that the cyanide when swallowed acts as a poison by reason of the production of prussic acid as a result of the action of the acid secretions of the stomach upon it. (Sur les Substances Toxiques, Paris, 1857, pp. 66, 103.) He carries this view so far that he believes if, in an animal out of health, the gastric juice were not secreted in its normal state of acidity, the cyanide would exert no poisonous action on the body! As a poison, the cyanide is probably not much inferior in activity to corrosive sublimate, but it differs from this compound in not possessing any locally corrosive action.

Analysis.—When heated, it yields cyanogen and metallic mercury. It is soluble in water. The solution differs from that of corrosive sublimate in not being precipitated by potash. Mercury is readily obtained from it by deposition on copper, and, when heated with an acid, prussic acid is distilled over. It has no odor of prussic acid in solution.

TURBITH MINERAL. SUBSULPHATE OF MERCURY.

Symptoms and appearances.—Fatal cases of poisoning by this compound are by no means common. Although insoluble in water, it is undoubtedly a strong irritant poison, and is capable of causing death in a comparatively small dose. A well-marked instance of its fatal operation was communicated to the Pathological Society, by Mr. Ward, in March, 1847. A boy, æt. 16, swallowed *one drachm* of this preparation. It produced a burning sensation in the mouth and throat, and vomiting in ten minutes. In about an hour there was paleness with anxiety of countenance, coldness of surface, constant sickness, sense of heat and constriction in the throat, and burning pain in the stomach with cramps. The irritability of the stomach continued in spite of treatment, and after two days there was salivation with mercurial fœtor. The gums acquired a deep bluish tint, and began to ulcerate. The patient died in about a week after he had taken the poison, without convulsions, and without suffering at any period from symptoms of cerebral disturbance. The principal *appearances* were: inflammation of the gullet,—its mucous membrane at the lower part peeling off; the inner surface of the stomach near the cardia and pylorus was covered with bloody spots; the small intestines were contracted, the inner coat reddened, and petechial spots were found upon it, but chiefly in the large intestines. The parotid and submaxillary glands were swollen. Mercury was detected in the intestines (see Med. Gaz. vol. xxxix, p. 474). From this account it will be perceived that the turbith mineral produces effects somewhat similar to those of corrosive sublimate, but it is less active.

Analysis.—Turbith mineral is a heavy powder of a yellow color, becoming of a dark olive by exposure to light. It is scarcely soluble in water, but has a strong metallic taste. When heated in a tube, with or without carbonate of soda, it yields metallic mercury. It may be analyzed by boiling it in potash, in which case sulphate of potash and peroxide of mercury result—the acid and the base are then easily determined.

NITRATES OF MERCURY.

Symptoms and effects.—These are corrosive poisons which are used for various purposes in the arts. They are solid white salts, soluble in cold water, if there be a little excess of acid present. The acid pernitrate caused death in a case reported by Mr. Bigsby, in the Medical Gazette (vol. vi, p. 329). A butcher's boy dissolved some mercury in strong nitric acid, and swallowed about a teaspoonful of the solution. Soon afterwards he suffered excruciating pain in the throat, gullet, and stomach: there was great anxiety, with cold skin, small pulse, colic, and purging. He became gradually weaker, and died in about two hours and a half. On inspection, the throat, gullet, and stomach were found corroded and inflamed. Although he survived so short a time, the mucous membrane of the stomach was of a deep red color. I have elsewhere related a case in which the application of the pernitrate of mercury to the throat as an escharotic caused immediate death by asphyxia. (See Guy's Hosp. Reports, Oct. 1850, 206.) The acid nitrate of mercury has often been employed by accoucheurs as a local application in diseases of the neck of the uterus. In one instance in which it was thus used, the ordinary symptoms of mercurial poisoning showed themselves, and the patient appears to have suffered severely. (Medical Gazette, vol. xlv, p. 1025.)

At the Leicester Summer Assizes, 1857, a girl was charged with administering nitrate of mercury to her mistress (Reg. v. E. Smith). The evidence showed that the accused had put the poison into some camomile tea prepared for the prosecutrix. Only a small quantity was taken, as the tea had a nauseous taste. The symptoms were: a burning sensation in the throat and stomach, violent vomiting with severe pain in the abdomen. By some extraordinary blunder, the girl was indicted under the statute which makes it penal to cast or throw or apply to any person any corrosive fluid, &c.: and although the words "cause to be taken" are introduced, Cresswell J. ruled that the statute implied, *external* and not internal administration! As the indictment was wrongly laid, the accused was acquitted. A report of other medico-legal cases will be found in the Ann. d'Hyg. Juillet, 1842; and Journal de Chimie, 1846, p. 734.

Analysis.—In the solid state, the crystals, when heated in a tube, yield nitrous acid vapor,—peroxide of mercury, and globules of metallic mercury: when heated with carbonate of soda, metallic mercury is easily obtained. The *solution* possesses all the properties of corrosive sublimate, so far as the tests for mercury are concerned (ante, p. 415);

but it gives no precipitate with nitrate of silver. When copper is immersed in it, mercury is deposited on the metal, and nitrate of copper is formed.

CHAPTER XXVIII.

ON POISONING BY LEAD—ACTION OF THE METAL—POISONOUS SALTS OF LEAD—THE ACETATE AND CARBONATE—SYMPTOMS OF ACUTE POISONING BY SUGAR OF LEAD AND GOULARD'S EXTRACT—EFFECTS PRODUCED BY CARBONATE OF LEAD—CHLORIDE, NITRATE, AND SULPHATE—CHRONIC POISONING—VARIOUS MODES OF—SYMPTOMS—EFFECTS OF EXTERNAL APPLICATION—APPEARANCES AFTER DEATH—FATAL DOSE—PERIOD OF DEATH—TREATMENT OF ACUTE AND CHRONIC POISONING.

General remarks.—Lead appears to exert no poisonous action when swallowed in the metallic state. Under exposure to air, water, and certain acids, it is easily converted to a poisonous salt—carbonate of lead. In the interior of the body, however, we have no reason to believe that this metal produces any noxious effects. Bullets and shot are occasionally swallowed without giving rise to symptoms of poisoning. In a case that occurred to Dr. Davis, a boy, æt. 4, swallowed a leaden bullet. The child suffered no inconvenience after the bullet had reached the stomach. It was passed by the bowels in about a week, much roughened on the surface, and, by comparison with a bullet from the same mould, it had lost ten grains. (Med. Gaz. vol. xxxviii, p. 686.) A man, æt. 28, swallowed in three days three ounces of small shot (No. 4). This is an alloy of lead and arsenic, the latter metal being in very small proportion. On the third day there was great anxiety and depression, with sunken features, coldness of skin, dizziness, and numbness in the arms and legs. He continued getting worse in spite of treatment; his bowels were obstinately torpid, and there was increased numbness in the arms, and dizziness. Purgatives were given, the alvine discharges were examined, but only one pellet was found; so that if he passed the shot at all, it must have happened in the three days before he was seen by Dr. Bryce. This man perfectly recovered in a fortnight. (Lancet, Dec. 31, 1842.) As these are the symptoms produced by the oxide or a salt of lead, it is probable that the metal was partially oxidized and converted into a salt by the acid mucous secretions of the stomach. It was then absorbed, and produced its usual effects. The handling of metallic lead or pewter has been known to produce the effects of chronic poisoning. In this case, probably, the lead is also oxidized; and where there is a want of cleanliness, it is brought to a state favorable for absorption by the chemical action of the secretions of the skin upon it.

It was formerly thought that the only poisonous salt of lead was the carbonate; but it is now known that it is the oxide of lead which produces the effects, and that every salt of lead is poisonous, provided it is in a state fitted for absorption, either by the skin or the mucous membrane of the stomach. Dr. C. G. Mitscherlich found, by experiments on animals, that the acetate of lead mixed with acetic acid is more energetic than when given in the neutral state. (Brit. and For. Med. Review, No. vii, p. 208.) The only two salts of this metal which require special notice, in a medico-legal point of view, are sugar of lead and white lead.

SUGAR OF LEAD. ACETATE. SUBACETATE. GOULARD'S EXTRACT.

SYMPTOMS.—ACUTE POISONING.—Acetate or sugar of lead is commonly met with in solid heavy crystalline masses, white, or of a brownish-white color. It resembles loaf-sugar in appearance, and has been mistaken for it. It is retailed to the public at the rate of from three-halfpence to two pence an ounce; and for quantities less than this, one penny is charged. It has a sweet taste, which is succeeded by an astringent or metallic taste. It is very soluble in water. Four parts of water at 60° will dissolve one part; and it is much more soluble at a boiling temperature. It is dissolved by alcohol.

Sugar of lead is by no means an active poison, although it is popularly considered to possess a virulent action. In medical practice, it has often been given in considerable doses without any serious effects resulting. Dr. Christison states that he has given it medicinally, in divided doses, to the amount of eighteen grains daily for eight or ten days, without remarking any unpleasant symptom whatever, except, once or twice, slight colic. (Op. cit. 555.) When, however, the quantity has been from one to two ounces, the following symptoms have been observed: a burning, pricking sensation in the throat, with dryness and thirst; vomiting supervenes; there is uneasiness in the stomach, sometimes followed by violent colic. The abdomen is tense, and the parietes have been occasionally drawn in. The pain is relieved by pressure, and has intermissions. There is in general constipation of the bowels. If any fæces be passed, they are commonly of a dark color, indicative of the conversion of lead to sulphuret. The skin is cold, and there is great prostration of strength. When the case is protracted, the patient has been observed to suffer from cramp in the calves of the legs, pain in the inside of the thighs, numbness, and sometimes paralysis of the extremities, with other symptoms of chronic poisoning. The affection of the nervous system is otherwise indicated, by giddiness, stupor, and even coma. A well-marked blue line has been, in some cases, observed round the margin of the gums, where they join the teeth; but this is chiefly noticed when the case is protracted.

The symptoms of poisoning by this salt are subject to variation, but not to the same degree as in other cases of irritant poisoning. Colic and constipation of the bowels are generally met with, and the vomiting is commonly not very violent; it requires to be promoted by the use of emetics. A woman, æt. 41, was admitted into Guy's Hospital (May, 1846). It was ascertained that two hours previously she had swallowed about one ounce and a half or half a teacupful of sugar of lead, dissolved in some water. She experienced a nauseous metallic taste in her mouth, with a burning heat in the mouth, throat, and stomach. She took some water to remove the taste. This made her vomit. Her mouth was very dry; she had great pain at the pit of the stomach; and in two hours after the poison had been taken, she felt sleepy and stupid, alternately perspiring and shivering. She complained of a violent twisting pain in the abdomen, which was relieved by pressure; with this there was a sensation of sickness. She felt weak and languid; complained of cramp in the thighs, and numbness all over the body, with giddiness. The gums felt to the patient to be in lumps, and they were very tender: the breath was foul. The pulse was hurried, and the tongue coated; countenance anxious and excited; skin dry, cold and hot alternately. The urine was passed freely. The next day there were pains all over the body, with numbness and sickness. On the third day she was very sleepy, but in less pain. For several days the abdomen was painful on the slightest pressure. She left the hospital in five days.

In March, 1858, a young man swallowed an ounce of acetate of lead in half a tumbler of water. He took what was undissolved as well as the portion dissolved. In a quarter of an hour he vomited; there was pain in the forehead, with severe pain in the abdomen; and in three quarters of an hour from the time of swallowing the poison, he was purged once freely. When brought to the Northern Hospital two hours subsequently, the only symptom was in the abdomen. The pulse presented nothing remarkable. The next day the bowels were constipated, and sulphate of magnesia was given. He was discharged well the day following. (Med. Times and Gazette, March 20, 1858, p. 296.)

When the patient recovers from the first symptoms, the secondary effects often last for a considerable time. In two cases which occurred to Mr. Gorringe, two girls swallowed an ounce of the sugar of lead by mistake. Soon afterwards they felt a burning pain in the mouth, throat, and stomach, and in a quarter of an hour they vomited freely; in half an hour there was severe pain in the bowels, with purging. Under treatment, recovery took place. (Prov. Med. Journ. April, 1846.) After a year had elapsed, they both suffered from severe pain in the stomach, which was tender on pressure. Nothing could be retained on the stomach; and there was a choking sensation in the throat, with some constitutional symptoms. A girl who had swallowed sixty grains of acetate of lead, and suffered severely from the primary symptoms, recovered and left the hospital in about three weeks, without any paralysis or other disorder affecting the muscular system. (Lancet, April 4, 1846, p. 384.) In another case, a woman, æt. 20, took one ounce of sugar of lead. She was placed under treatment at St. Thomas's Hospital. The chief symptoms were slight excoriation of the gums, a sensation of heat in the throat, and relaxation of the bowels. There was pain in the calves of the legs and thighs, with great thirst and restlessness. In six days the woman had quite recovered. (Med. Gaz. vol. v, p. 704.) Large doses given medicinally are not always borne with impunity. Dr. Joynes prescribed thirty grains of the acetate in four days for inveterate diarrhœa. The medicine appeared to be of service; but in a week afterwards the patient was seized with pain in the stomach; a severe attack of lead colic came on, and continued for eight days. (Harrison on Lead Poison, p. 148; see also Provincial Transactions, vol. i, p. 119.)

The symptoms are sometimes slow in appearing. The following case occurred to Dr. Hviding. A girl swallowed about three drachms of the acetate of lead in broth. It was not until *two hours* afterwards that she began to experience sharp colicky pains in the abdomen, followed by vomiting. No medical treatment was employed for three days; and the only marked symptom then was obstinate constipation. Doses of castor oil were prescribed, and the girl recovered. (Journal de Chimie, 1845, 256.)

A series of cases of poisoning by acetate of lead has been reported by Mr. Bancks, of Stourbridge. (Lancet, May 5, 1849, p. 478.) By some accident, about thirty pounds of this substance were mixed at a miller's with eighty sacks of flour, and the whole was made into bread by the bakers, and supplied as usual to their customers. It seems that no fewer than 500 persons were attacked with symptoms of poisoning after partaking of this bread. In a few days they complained of a sense of constriction in the throat and at the pit of the stomach, violent crampy pains round the navel, rigidity of the abdominal muscles, a dragging pain in the loins, and cramp, with paralysis of the lower extremities. There was obstinate constipation, and the urine was scanty, and of a deep red color. The pulse generally was slow and feeble; the countenance anxious and sunken, frequently of a peculiar livid hue; tongue flabby; gums marked by a deep blue line. The surface was cool, and there was a general arrest of the secretions. Sickness was not a uniform symptom; and even when it existed at first, it speedily subsided. The mental faculties were undisturbed. Not one of the cases proved fatal; but among the more aggravated, there was great prostration, with collapse, livid countenance, universal cramps, numbness, and other alarming symptoms. After apparent convalescence, some of the symptoms returned in a more aggravated form, without any obvious cause, and for a long time the patients were out of health. Inflammation was not observed. Purgative medicines were found most effectual in the treatment. The quantity of acetate of lead taken by each person could not be determined, as, on analysis, the samples of bread were found to be very unequally impregnated with the poison.

Goulard's extract is generally seen under the form of a reddish-colored liquid, as it is often made with common vinegar instead of acetic acid. *Goulard water* is a lotion compounded of from one drachm to one drachm and a half of Goulard's extract, or solution of subacetate of lead, of a drachm of spirit, and a pint of water. It is not poisonous unless administered at intervals in small doses; it may then cause chronic poisoning. Goulard's extract, or subacetate of lead, has caused death in at least four instances,—one in France and three in England. The symptoms produced are similar to those above described. The subacetate is much more powerful as a poison than the neutral acetate, probably from its containing a larger quantity of the oxide of lead. One fatal case of poisoning by Goulard's extract is recorded in the Coroner's Return for 1837–8. In January, 1840, two other cases of poisoning by it occurred in London in two children, aged respectively four and six years. The quantity taken by the children could not have been great, but they both died within thirty-six hours. The symptoms were at first violent vomiting and purging;—in one case they resembled those of Asiatic cholera. Dr. Hall states that he gave ninety-six grains of this compound in three days—thirty-two grains in the first four hours—and the only unpleasant effect was pain in the bowels. This was in a case of spitting of blood. (Harrison on Lead Poison, p. 145.) These results must not be considered paradoxical. They are, to a great extent, explained by reference to the influence of disease on medicinal and poisonous agents (see ante, p. 98). Dr. Hall did not take this quantity himself, nor try it on a healthy person, or the results might have been different.

The *chloride* and *nitrate* of lead are poisons, but not of an active kind. Dr. Christison found that it required 400 grains of the crystallized nitrate to kill a dog in sixteen hours. (On Poisons, p. 549.) Some years since, a woman, who had swallowed an unknown quantity of chloride of lead, was brought to Guy's Hospital. The only urgent symptom was vomiting. She recovered and left the hospital on the same day.

Carbonate of lead, known also as *white lead*, *ceruse*, or *Kremser white*, is commonly in the fórm of heavy white masses resembling chalk. It is insoluble in water, but in large doses it posesses poisonous properties. In October, 1844, the late Dr. Snow met with the following case of poisoning by this substance. A child, æt. 5, ate a portion not so large as a marble, ground up with oil. For three days he merely suffered from pain in the abdomen and costiveness. On the third night, the child became rapidly worse, and there was vomiting. He died ninety hours after taking the poison, having passed some offensive motions of a greenish-black color (probably from sulphuret of lead) before he died. It is remarkable that in this case so small a quantity should have proved fatal without exciting any marked symptoms of irritation in the first instance. There are many cases of poisoning by the carbonate of lead in the human subject; but it has in these instances proved insidiously fatal, by inducing colica pictonum. The following case of recovery from a large dose of carbonate of lead is reported by Mr. Cross. A woman, æt. 33, took, by mistake for a dose of magnesia, from *six to eight drachms* of carbonate of lead. Five hours afterwards, she was seen by her medical attendant. She was in a cold perspiration, breathing heavily, constantly vomiting, her pulse hard, small, and quick. There was great anxiety of countenance, with dryness of the throat, and a sense of heat in the stomach, with painful colics. Castor oil and sulphate of magnesia, with diluted sulphuric acid, were given to her,—the last at frequent intervals. The extensor muscles became paralyzed, and the flexors rigidly contracted; the colics were so excruciating, that the patient generally fainted after each paroxysm. The evacuations from the bowels were of a dark color. The symptoms abated; but the next day there was nausea and faintness, with griping pains. In four days she was convalescent. A somewhat similar case is reported in Casper's Wochenschrift for 1844, in which a man, æt. 20, swallowed, by mistake for chalk, between five and six drachms of carbonate of lead. In a few hours it produced all the symptoms of irritant poisoning, thirst, burning pain, and incessant vomiting; yet, although he was not seen for twenty-four hours after taking the poison, he perfectly recovered in the course of a short time under very simple treatment. His recovery was probably due to the greater part of the carbonate having been ejected by the early vomiting. (Ann.

d'Hyg. 1845, ii, 226.) These cases show that the carbonate of lead, although poisonous, is not very energetic. Its action as a poison is probably not greater than that of the acetate; and, so far as observations on the human subject extend, it is less active than the subacetate.

A case is related in the Annales d'Hygiène (April, 1844), which shows that shot used in cleaning bottles may be chemically acted on by the acids of the wine or liquid, and give rise to the production of this poisonous salt of lead. A person, after having swallowed a few glasses of liqueur, suffered from the most violent colicky pains, and all the symptoms of irritant poisoning. Dr. Hanle, who was immediately called, having observed that the liquor remaining in the bottle was very turbid, poured it off for analysis, when he found firmly wedged in at the bottom of the bottle, ten leaden pellets, which had become so completely transformed to carbonate of lead, that there was only a small nucleus of the metal left. So long as the liquor was clear, no accident had arisen from its use; but the symptoms of poisoning appeared immediately when the turbid portion at the bottom of the bottle, containing the salt of lead either suspended or dissolved, was swallowed.

The *sulphate of lead*, by reason of its insolubility, is commonly regarded as inert, and the results of the following experiments appear to justify the opinion. M. Dupasquier ascertained that *seventy-seven* grains of the sulphate might be given to a dog, kept fasting for twenty-four hours, without exciting vomiting or any other unpleasant symptom. The dog was kept four days, and the dose produced no effect. On killing the animal and inspecting the body, there were no abnormal appearances. Doses of 150 and 300 grains were given to other dogs, without producing symptoms of poisoning. (Consult Méd. Lég. 1848, p. 15[illegible] Orfila states that he gave to a dog 554 grains in a finely pulverised state, without any injurious effects resulting. The dog ate its food as usual on the following day. (Op. cit. i, 690.)

The chromate and iodide of lead are ranked among lead poisons. Of poisoning by the former substance, there is not, so far as I know, any instance recorded; and with respect to the latter, there is a single experiment on a cat recorded by M. Paton, from which we learn that nineteen grains in two doses produced paralysis of the hinder legs, and apparently colicky pains. The animal died in three days. (Orfila, Toxicol. i, 702.)

The oxides of lead, *litharge* and *red lead*, are known only as producing the effects of chronic poisoning in the working of certain trades, and by accidental admixture with food.

Chronic poisoning.—The effects of chronic poisoning are more frequently witnessed in reference to the salts of lead than of other metallic irritants. Any salt of lead taken in small doses at intervals, may give rise to chronic poisoning, producing lead-palsy or other forms of lead disease. White lead and litharge are the compounds to which chronic poisoning may be most frequently traced. The medicinal use of the acetate, if continued for an undue length of time, may so saturate the system with the metal as to occasion this form of poisoning. A child, æt. 6, took, in a quack medicine, 1-15th of a grain of acetate of lead two or three times a day for nearly nine weeks. It was then found to be laboring under symptoms of poisoning by lead, and two days afterwards the child died. The first effects of taking the medicine were, that the child fell away, and complained of colicky pains; the bowels were constipated, the evacuations when passed, black and offensive, and there was fœtor of the breath. Latterly the child was drowsy and the limbs were paralyzed. Upon the day of its death it was convulsed, and shortly before death it fell into a state of coma. (Pharmaceutical Journal, Dec. 1845, p. 259. Case by Dr. Letheby.) This case shows that more injury may be done by frequently repeated small doses than by one or two large doses. The child took in nine weeks no more than Dr. Christison prescribed, without any injurious effects, in the space of two days.

The disease called *colica pictonum* or *painter's colic* derives its name from its supposed seat in the colon, but according to some observers, the seat of pain is in the muscular coverings of the abdomen. (Dr. Briquet, Archives Générales, Feb. and March, 1838; and Dub. Hosp. Gazette, Aug. 1858, p. 237.) This disease may be regarded as a *chronic* form of poisoning by carbonate of lead. The carbonate finds its way into the system, among white-lead manufacturers, either through the skin or through the lungs, or both together;—it is diffused in a fine atmosphere, and is thus respired. It has been remarked, that in factories where the powder was dry, not only have the laborers suffered, but horses, dogs, and even rats, have died from its effects. Since the practice has arisen of grinding the carbonate in water, cases of colica pictonum have not been so numerous. They are still, however, not unfrequent among painters, plumbers, pewterers, the manufacturers of some kinds of glazed cards, the bleachers of Brussels lace, and among those engaged in the glazing of pottery, when oxide of lead is employed in the glaze. In ten years, according to Dr. Clemens, there were 1898 cases of chronic poisoning by lead among workmen, admitted into the hospitals of Paris. (Casper's Viertelj. 1853, ii, p. 177.) Out of 1330 cases received during five years (1838–42) in the Parisian hospitals, 655 were among the workers in white lead and painters. Of 341 who were workers in white lead, 55 died. The workers in metals,—plumbers who handle metallic lead, are but little subject to the disease. Only 22 cases of this kind occurred in the five years. (Gaz. Med. Janvier 17, 1847.)

The workmen who are employed to whiten Brussels lace by beating white lead into the fibre, constantly breathe an atmosphere of this poisonous salt, and suffer, according to M. Chevallier, from dryness of the fauces, colic, and other symptoms of chronic poisoning by lead. (Ann. d'Hyg. 1847, i, 111, 1855, ii, 317.) It becomes a question whether those females who wear this lace in close contact with the skin, may not suffer from symptoms of lead-poisoning. At any rate, those who prepare the lace, suffer and die from the effects of lead-poisoning. A fatal case of this kind is reported in the Med. Times and Gazette. (Dec. 19, 1857, p. 636.) Lead was found in the viscera of a female who had thus fallen a victim to lace-whitening. (Gazette Médicale, Dec. 11, 1847, p. 993; and Ann. d'Hyg. 1856, ii, 316.) Actors who employ carbonate of lead as a cosmetic, to give paleness to the countenance, are liable to attacks of lead-colic. (Med. Times and Gazette, August, 1852, p. 228.) The makers of glazed cards, in which white lead is largely employed, also suffer from this disease. So easily is the system affected, that colic and paralysis have been known to arise from a person working or sleeping in a recently-painted room. (Lancet, Oct. 26, 1844.) In a case reported by Dr. Chowne, a man who slept in a newly-painted room for a few nights was attacked with paralysis. (Med. Gaz. xxxix, 255.) In these instances, the noxious emanations are received through the lungs. Dr. Alderson mentions several cases of a similar kind, and he calls this form acute paralysis from lead. (Lancet, Oct. 30, 1852, p. 391.) I have myself suffered from severe colic by respiring the vapor of fresh paint. It is not improbable that, in these cases, the carbonate of lead is carried off in vapor, in combination with that of the essential oil of turpentine. There are numerous other cases in which lead, or its preparations, by mere contact with the skin, have been known to produce the usual results of lead-poisoning. The late Mr. Scanlan communicated to me a case in which an infant was paralyzed by reason of its having been washed with water containing a finely-diffused oxide and carbonate of lead. Dr. Todd mentions the case of a man in King's College Hospital, who suffered from lead-palsy. He had been a potman, and the palsy was attributed to the constant handling and cleaning of pewter pots. (Med. Gaz. xlviii, p. 1047.) In the Journal de Chimie (Juillet, 1858, p. 434), the case of a compositor is related, from which it appears, that local paralysis of the right hand was induced in a week as the result of handling new type. The sharp edges of the type produced abrasions of the thumb, fore and middle fingers,—a condition which favored absorption. In five days the wrist became progressively weaker: in a week the hand dropped so that it could not be raised, and there was loss of power to grasp any article with firmness. There was a faint blue line at the edge of the gums. This appears to have been an instance of purely local action affecting only one hand, and not preceded by colic. It was found that in this case baths of the sulphuret of potassium effected a cure. Cosmetics and hair-dyes containing preparations of lead, may also produce dangerous effects. I have met with an instance, in which paralysis of the muscles on one side of the neck arose from the imprudent use of a hair-dye containing litharge.

The *symptoms* of chronic poisoning by lead are well marked. There is first pain, with a sense of sinking, commonly in or about the region of the navel—the seat of the colon. Next to pain there is obstinate constipation, retraction of the abdominal parietes, loss of appetite, thirst, dryness of the mouth and throat, fœtid odor of the breath, sallowness of the countenance, and general emaciation. The skin is dry, acquires a yellowish or earthy color, and the patient experiences a saccharine, metallic, or astringent taste in the mouth. A symptom of a peculiar nature has been pointed out by the late Dr. Burton (Med. Gaz. xxv, 687), namely, a *blueness* of the edges of the *gums*, where these join the bodies of the teeth: the teeth are of a brownish color. This coloring of the gums has been so frequently observed, that most pathologists now regard it as a well-marked, although not a certain, indication of lead-poisoning. A similar blue mark round the edges of the gums has been noticed in other cases of poisoning—as by mercurial preparations, and as a result of the medicinal administration of the salts of silver. On the other hand, in certain cases of chronic poisoning by lead it may be absent (see a case by Mr. Fletcher, Med. Times, Feb. 14, 1846, p. 395); as where, for example, the individual has ceased to expose himself to emanations of lead. Dr. Thomson, of Stratford-upon-Avon, has observed, with respect to this coloring of the gums, that it was absent in a case of chronic lead-poisoning in which the other symptoms were well marked. In another instance, in which the poison was derived from the same source, there was no paralysis of the hands, but the gums were deeply tinged. He has also seen this blue tinge of the gums in painters who had not suffered from any of the constitutional effects of the poison. (Med. Times, Dec. 1848, p. 195.) In some cases in which the blue color has not appeared, the gums have presented a fungous appearance, and have been observed to bleed frequently. (Med. Times and Gaz. Jan. 30, 1858, p. 124.) Hence, while a blue line indicates poisoning by lead, its absence is not to be taken as a proof that this poison is not in the system. In April, 1846, a woman was admitted into Guy's Hospital, with some obscure symptoms of chronic gastritis, and dull aching pains in the stomach and back. For ten weeks previously, her bowels had only acted under the use of medicine. On the third day after her admission, a distinctly blue line was for the first time noticed on both gums, and there was trembling of the hands with paralysis of the extensors of the wrists. She became insensible, and died a week after her admission. An analysis of the substance of the liver showed that this organ contained a small portion of lead, and, although no evidence could be obtained that deceased had taken the poison, the chemical analysis confirmed the opinion derived from the symptoms, that this was a case of lead-poisoning. (Guy's Hosp. Reports, 1846, p. 471.)

It is not improbable, that many cases of supposed cerebral, spinal, or heart disease, are really due to the insidious and unsuspected action of lead upon the system. In December, 1856, a case fell under the notice of my colleague, Dr. Addison,—that of a pale, exsanguined man, who had suffered from head symptoms, failing power in the limbs, irregular pains in the stomach, and palpitation of the heart. In looking at his tongue, it was observed that there was a blue line on the gums, and on further inquiry, it turned out that the man had been a worker in red lead, and had suffered much from constipation. The arms had been more affected by the loss of power than the legs—one of the peculiar features of lead-palsy. The case was thus recognized and treated as one of lead-poisoning:—the blue line on the gums having first led to a suspicion of its real character. (Med. Times and Gaz. Dec. 27, 1856, p. 643.)

The earliest period at which this blue line on the gums first shows itself is not known; it is the result of a slow deposit of absorbed lead, and in an early stage, unless specially looked for, may escape notice. According to some authorities, the period at which the line is first seen, varies with the quantity of lead taken. Dr. Burton states, that the discoloration has been produced in twenty-four hours, by giving four doses of five grains each of the acetate of lead in six hours; and he thinks it might be obvious in four or six hours when large doses are given. (Med. Chir. Trans. xxiii, p. 78; and Harrison on Lead-poisoning, p. 59.)

When once produced, this appearance is very persistent. Dr. Thomson noticed it in the case of a patient poisoned by drinking water containing lead, about a year previously; and in a severe case of colica pictonum with slight paralysis of the hands, occurring in a house-painter, after repeated attacks, a faint indication of it was recognized four years after the man had ceased to be subjected to the influence of the poison. In this instance, the color had, in great part, disappeared at the end of the first year. (Med. Times, Dec. 1848, p. 195; see also ante, p. 66.) Among the symptoms is a marked effect on the nervous system. There is a dull, numb feeling in the skin,—especially noticed in the fingers and fore-arms, trembling of the arms and legs, unsteadiness in walking or in any muscular exertion, with rheumatic pains in the loins. Symptoms affecting the brain also present themselves. The loss of power in the wrists is manifested chiefly by a paralysis of the extensor muscles, so that the hand drops. The body becomes emaciated, the legs œdematous, and the person dies exhausted.

A common cause of chronic poisoning by lead, is the use of *water* which has acquired an impregnation of a salt of lead from being kept in a cistern or pipe of this metal. The symptoms come on very insidiously, and are not materially different in their course and character from those above detailed. Dr. Thomson has well described them in the paper to which I have referred: they are manifested by pains in the stomach and bowels, constipation,—evacuations, when passed, dark-colored; headache; flushing of the face; pains in all the joints and limbs, but especially the wrists, with great depression of spirits; paralysis of the extensor muscles of the arms and hands; countenance dark and sallow; eyes sunk; tongue flabby, indented, and of a whitish livid color, with a dark blue line along the transparent edge of the gums. It is obvious that symptoms of this nature may be overlooked or referred to some other cause. In 1857, a man was admitted into Guy's Hospital, laboring under some of the symptoms above described. He had also a line of blue color at the edges of the gums. So far as it could be ascertained, he had been exposed to no cause of poisoning by lead. He lived at Norwood; and, on requiring for examination a sample of the water which he had been in the habit of drinking, it was found to be impregnated with lead derived from a leaden cistern. In these cases the cause is not commonly suspected until the symptoms are fully developed; and it is only after the patient has suffered for some time from the effects of the poison, that the symptoms assume the form of lead-colic. (Harrison on the Contamination of Water by Lead, 1852, p. 60.)

A remarkable series of cases presenting this form of chronic poisoning by lead, occurred in the members of the ex-royal family of France, at Claremont, in 1848. I have elsewhere referred to these cases (ante, p. 66), and a full account of the symptoms by Dr. De Mussy, will be found in the Dublin Quarterly Journal. (May, 1849, p. 405; Med. Gaz. xliv, p. 260; and Harrison on Lead Poison, p. 122.) Out of thirty-eight persons, using water impregnated with lead to the extent of about one grain in the imperial gallon, thirteen were attacked with symptoms of chronic poisoning, but presenting various shades of difference. There was a sallow complexion, yellowness of the eyes, wasting of the body, with frequent attacks of colic, nausea, and obstinate constipation. Among the nervous symptoms, there was great restlessness, depression, and, in some, an excessive sensibility of the skin. As confirmatory of the statement above made respecting the blue line on the gums (ante, p. 485), this symptom existed only in one-half of the patients, and there were others in whom the blue line existed who did not experience any inconvenience from the lead. Lead was not only found in the water in small proportion, but in the urine of some of the patients; showing that it was undergoing elimination by this secretion. It is remarkable that six children of the family, aged from 3 to 7 years, did not show any symptoms. In some of the patients Dr. De Mussy observed, in addition to the blue line on the gums, slate-colored spots on the mucous membrane of the inside of the mouth. In one case, the mucous membrane of the mouth and tongue of a worker in white-lead was observed to be entirely of a slate-blue color.

Effects of external application.—Some remarks have been already made on this subject (ante, p. 483). The application of any preparation of lead in powder, ointments, or diffused in water, is sufficient to excite all the symptoms of chronic poisoning. A nurse-maid was in the daily habit of bathing a healthy infant in the distilled water obtained from a leaden pipe connected with a steam-boiler. The child grew up almost paralytic and tottering in gait, and the cause was not suspected until the water was chemically examined and found to contain lead.

Most *hair-dyes* are composed of a mixture of lime and oxide or a subsalt of lead. (See Ann. d'Hyg. 1832, ii, 324.) The long-continued use of these preparations may give rise to symptoms for the origin of which a practitioner might not be able to account (see ante, p. 434). Dr. Brück, of Hanover, observed that a violent ophthalmia was induced in a lady who had used for dyeing her hair, a substance called *Poudre d'Italie*, which, on chemical analysis, was found to consist of lead and lime. (Med. Gaz. Nov. 1842.) The facts connected with poisoning by lead or its preparations, applied *externally*, are, therefore, of some interest. Some of these have been already noticed under the head of chronic poisoning. The pure metal frequently handled may, unless strict cleanliness be observed, produce the effects of poisoning (ante, p. 484). Among the cases mentioned by Orfila is one of a female, who was in the habit of applying, for a long period, to her face and neck, a cosmetic containing a preparation of lead. After six months there were the usual symptoms of chronic poisoning. This female ultimately became blind and paralytic, and soon afterwards died. In another instance, the symptoms had become so firmly established before the cause was suspected, that no treatment sufficed to relieve them. (Toxicologie, i, 680.) I have above referred to a case of a similar kind within my own observation, which did not prove fatal. Notwithstanding these facts, M. Tanquerel does not consider that serious symptoms can be produced by preparations of lead coming in contact with the unbroken skin. If the skin be abraded, then absorption may take place rapidly. A case is reported by Taufflieb, in which the frequent application of lead-plaster to an ulcer of the leg, was followed in less than three months by all the symptoms of chronic poisoning. (Galtier, i, 698.) The use of a simple lead-wash in cutaneous disorders has not been attended with any injurious effects; but the application of white-lead and linseed oil to an abraded surface, produced in one instance an attack of lead-colic. (Beck's Med. Jur. vol. ii, p. 650.)

Appearances after death.—The appearances observed in cases of acute poisoning are very characteristic. The mucous membrane of the stomach and intestines is covered with thick white or whitish-yellow layers of mucus mixed with the salt of lead, and beneath this, the membrane is reddened or ecchymosed. In the fatal cases of poisoning by subacetate of lead (ante, p. 430), the following appearances were found. The mucous membrane of the stomach was of a gray color, but otherwise perfectly healthy. The intestines were found much contracted, in one instance more so than in the other. A case is reported by Orfila, in which an inspection was made of the body of a man who had been killed by taking a quantity of Goulard's extract. He died within forty-eight hours, and there was well-marked inflammation of the alimentary canal from the œsophagus downwards. The villous coat of the stomach was completely softened, and the effused mucus was found to contain the poison. (Toxicologie, i, 671.)

In a case related by Dr. Kerchoffs, the mucous membrane of the stomach was found abraded in several places, especially near the pylorus; and most of the abdominal viscera were in a state of high inflammation. A trial for murder by this substance took place at the Central Criminal Court, in November, 1844 (Reg. v. Edwards). In this case the stomach and intestines are stated to have been found inflamed, and there were dark spots on the former. In animals, according to Dr. Mitscherlich, when the dose is large, the mucous coat of the stomach is attacked and corroded; this change appears to be purely chemical, and takes place in all the organs of the body with which the salt of lead comes in contact. If given in a small dose, it is decomposed by the gastric secretions, and exerts no corrosive power on the mucous membrane. When the acetate of lead was given in a state of albuminate dissolved by acetic acid, death took place with great rapidity; and, on inspection, the stomach was not found to be corroded. This corrosive action belongs to the neutral salt, and is not manifested when the dose is small, or when the poison is combined with an acid. In the case of poisoning by the carbonate of lead, which proved fatal in ninety hours (ante, p. 431), the mucous membrane of the stomach was much inflamed and of a dark red color throughout.

In the *chronic* form of poisoning, the appearances are less distinct. The blue line on the gums may or may not exist in the dead body, according to circumstances. In the case of the female elsewhere related (ante, p. 435), there was thickening and enlargement of the coats of the stomach, and the mucous membrane was especially thickened. The large intestines were irregularly contracted and distended. There were spots of congestion upon the mucous coat, and the salivary glands were enlarged. In Dr. Letheby's case (ante, p. 438), the skin was of a dingy yellow, and the gums were of a deep blue color. The lungs were slightly congested, and there was an effusion of serum in the pleuræ. The blood was black and liquid. The stomach and intestines were pale and nearly empty,—the former contained half an ounce of a thick brownish fluid, in which lead was detected; the latter were contracted in some places and distended in others, and they presented several points of intussusception. The large intestines were in a similar condition. The bronchial and mesenteric glands were enlarged. The bladder and ureters were full of urine;—the rest of the viscera healthy.

Böcker describes the mucous membrane of the stomach and intestines as congested and softened, presenting often a yellow, gray, brown, or black appearance. The coats of the intestines are thickened, and the canal is irregularly contracted. The lungs are congested,—the muscles are pale, wasted, and converted to a fibrous tissue. (Vergiftungen, 1857, p. 49.) For further information on this subject, I must refer the reader to the Traité des Maladies de Plomb, par Tanquerel des Planches, 1839; Traité Pratique de la Colique de Plomb, par J. L. Brachet, 1850; and a translation of the work of Tanquerel des Planches, by Dr. Dana, U. S. 1848.

Fatal dose. Period of death.—Nothing is accurately known concerning the fatal dose of sugar of lead, or of carbonate of lead. The facts already detailed show that either substance may be taken in a comparatively large quantity, without producing serious effects. Thirty and forty grains of the acetate have been given daily, in divided doses, without injury in every case. The following additional cases, in some of which recovery took place under very disadvantageous circumstances, prove that sugar of lead is not an active poison. Dr. Iliff met with an instance where *an ounce* was swallowed in solution. The symptoms were pains in the abdomen resembling colic, with vomiting, rigidity, and numbness. It was three hours before any remedies were used, and five hours before the stomach-pump was employed. The person recovered. In a second case an ounce was swallowed: sulphate of magnesia was freely exhibited;—the stomach-pump was used, and the patient recovered. In October, 1835, a girl, æt. 19, dissolved about an ounce of acetate of lead in a cupful of water, and swallowed it. In a quarter of an hour violent vomiting came on, and she was taken to the North London Hospital. Sulphate of magnesia and diluted sulphuric acid were given to her. There was slight pain in the abdomen, weight in the head, dimness of sight, with pains shooting through the eyeballs. The abdomen was tender on pressure for several days; but in five days the patient was discharged cured. The fourth case occurred in Paris, in 1840. A girl swallowed *an ounce* of sugar of lead: the usual symptoms followed, and sulphate of soda was administered. She recovered. In a case reported by Dr. Evans, a woman recovered after having swallowed half an ounce of the acetate by mistake, under the free use of aromatic sulphuric acid. (Amer. Jour. Med. Science, Feb. 1847, 259.) Mr. Marshall mentions a case of recovery where two fluid ounces of Goulard's extract had been taken by mistake. (On Arsenic, p. 106.)

The dose of this poison required to destroy life was a subject of inquiry in Reg. v. Hume (Chelmsford Summer Assizes, 1847). The prisoner was charged with an attempt to murder her husband, by endeavoring to administer to him "a large quantity of a certain *deadly poison*, called sugar of lead!" According to the evidence, she made two boluses with flour and water, and the quantity of sugar of lead contained in them was equal to *twenty-six grains and a half.* This was pronounced to be sufficient to destroy life, although the grounds for this medical opinion do not appear. So far as I have been able to ascertain, there is not a single instance recorded in which even sixty grains have destroyed life. Van Swieten gave it to the amount of one drachm daily, for two days, before it caused any material symptoms. (See Christison, Op. cit. 555.) In another case, violent symptoms were produced by this dose; but the individual easily recovered from the effects. The observations and experiments of Orfila also prove that the vulgar belief of sugar of lead being an active poison, is erroneous. The fatal cases are so few in number, that it is impossible to fix, with any precision, the period within which this poison may destroy life.

TREATMENT.—This consists, in acute poisoning, in the free administration of solutions of the alkaline sulphates, either of soda or magnesia. The carbonate should be avoided, as the carbonate of lead is poisonous; while the sulphate is either inert, or possesses but very little activity. Purified animal charcoal has been recommended as an antidote, in consequence of the property which it possesses, to a certain extent, of separating oxide of lead from its saline combinations; but there is no record of its efficacy or utility.

An emetic of sulphate of zinc should be given if vomiting does not already exist; and castor oil, or croton oil, may be given, to promote free evacuation from the bowels. The stomach-pump may be occasionally employed with benefit. It is well known that albumen precipitates the oxide of lead when added in large quantity; and Mitscherlich has found that caseine, the albuminous principle of milk, is an effectual precipitant of the oxide of lead. Therefore it would be advisable to administer, in cases of poisoning by the soluble salts of lead, milk or albumen in large quantity. The compounds thus formed, as in the case of corrosive sublimate, may not be absolutely inert; but they are far less active than the acetate itself, and tend to prevent the action of the poison as a corrosive on the stomach. Six cases have been mentioned in which individuals recovered partly through treatment, after having swallowed one ounce of the acetate of lead. M. Bouchardat strongly advises the employment of the hydrated persulphuret of iron as a chemical antidote. This compound may be made by adding a persalt of iron to an alkaline persulphuret, the latter being in excess. It should be mixed with syrup, and preserved closely bottled to prevent chemical change. It is said to be inert, and may be given in large quantity. (Annuaire de Thérapeutique, 1847, p. 297.) The urine should be chemically examined for the purpose of tracing the disappearance of the poison from the body.

As a chemical antidote in poisoning by carbonate of lead, a mixture of vinegar and sulphate of magnesia may be employed.

In the treatment of chronic poisoning, the principal object is to remove the poison from the stomach. In a case of poisoning by water impregnated with lead, another source of supply should be immediately provided. The use of dilute sulphuric acid internally, and the most scrupulous attention to cleanliness of the skin by frequent ablutions, have been found the best means of preventing and treating some forms of chronic poisoning. When the poison is once absorbed, nothing can be done beyond trusting to its elimination by the urine and other secretions. The use of iodide of potassium has been strongly recommended, on the ground of its dissolving the lead, and carrying it off by the kidneys; but there is no satisfactory evidence that it has in any case accelerated a cure. (Lancet, Dec. 3, 1853, p. 522.) There is probably no metal which is retained so long in the body as lead, when it has been once deposited in the tissues (see ante, p. 66). The use of sulphuretted waters, or alkaline sulphuretted baths, is, under these circumstances, just as inefficacious as the use of diluted sulphuric acid. (See Orfila, Op. cit. i, 686; Galtier, Op. cit. i, 676.) Dr. De Mussy employed sulphur-baths in the treatment of the royal family of France; but all that he effected was a blackening of the nails and skin by the production of the sulphuret of lead! MM. Legroux and Girard have, however, employed these sulphur-baths, and, as they believe, with benefit. In some lead-works at Marseilles, in 1858, fifty-two out of 260 work people were affected with chronic poisoning within a month. Croton oil was given frequently, and sulphur-baths were employed towards the termination of the case. Two or three weeks were required for internal and external depuration in these cases of lead poisoning. (Med. Times and Gaz. Jan. 30, 1858, p. 125.)

Other modes of treatment will be found described by the writers to whose works on chronic poisoning I have already referred.

CHAPTER XXIX.

CHEMICAL ANALYSIS OF THE SALTS OF LEAD—TESTS FOR THE OXIDE OF LEAD—OBJECTIONS—LEAD IN ORGANIC MIXTURES—QUANTITATIVE ANALYSIS—DETECTION OF LEAD IN THE TISSUES—ABSORPTION AND DEPOSITION—LEAD IN ARTICLES OF FOOD—WINE—VINEGAR—PORK—SUGAR—SNUFF—IMPARTED TO FOOD BY SPURIOUS TIN-FOIL—HAIR-DYES—COSMETIC POWDERS—CONTAMINATION OF WATER BY LEAD—POISONING OF CATTLE BY LEAD.

CHEMICAL ANALYSIS.

Acetate of lead as a solid.—1. If a portion of the powder be heated in a small reduction-tube, it melts, then becomes solid; again melts, acquiring a dark color, and gives off vapors of acetic acid; a black mass is left in the tube, consisting of carbon and reduced metallic lead. There is no metallic sublimate formed. 2. It is very soluble in water, even when cold; river or spring water is turned milky by it, chiefly from the presence of carbonic acid and sulphates. Goulard water is generally opaque for this reason. 3. A small portion of the powder placed in a saucer, containing a solution of iodide of potassium, acquires a fine yellow color. 4. When treated with caustic potash, it remains white. 5. By hydro-sulphuret of ammonia, or sulphuretted hydrogen water, it is turned black, in which respect it resembles the white salts of some other metals. 6. When the powder is boiled in a tube with diluted sulphuric acid, acetic acid, known by its odor and volatility, escapes. All these properties, taken together, prove that the salt is *acetate* of lead.

Acetate of lead in solution.—If acetate of lead be presented in a state of solution, or if the solid salt be dissolved in water for the purpose of making further examination, we should note the following points: 1. A small quantity, slowly evaporated on a slip of glass, will give white and opaque prismatic crystals, which are turned yellow by iodide of potassium, and black by hydrosulphuret of ammonia. The solution is said to be neutral; but it is generally slightly acid. 2. *Diluted sulphuric acid* produces an abundant white precipitate, soluble in hydrochloric acid and in a large excess of caustic potash. 3. It is precipitated of a bright yellow color by the *iodide of potassium;* the yellow iodide of lead is soluble in caustic potash and concentrated muriatic acid, forming colorless solutions. 4. *Hydrosulphuret of ammonia*, or a current of sulphuretted hydrogen gas, produces in acid and very dilute solutions, a deep brown-black precipitate. This effect is observed when less than the 100,000th part of the salt is dissolved. The potash solutions of the sulphate (2) and of the iodide (3) are precipitated black by these tests. 5. Place a few drops of the solution in a clean platina capsule,—acidulate it with acetic acid, then apply, through the solution, to the surface of the platina, a thin polished slip of zinc: bright crystals of metallic lead are instantly deposited on the zinc. Although this is not a delicate mode of testing, yet by it the metal may be obtained from a small quantity of salt.

Among these tests, there is none so efficacious or certain as the sulphuretted hydrogen gas. A current of this, when properly applied, will reveal, by the production of a brown tinge, a quarter of a grain of a salt of lead, in a gallon of water, *i. e.* about 1-300,000th part. Sulphuric acid failed to indicate 1-15th of a grain of the acetate in an ounce of water, and began to form a decided precipitate only when the quantity amounted to one-fifth of a grain. Iodide of potassium failed to act when the salt of lead was much diluted. It did not indicate the presence of half a grain in twelve ounces of water. The fifteenth part of a grain, which was clearly indicated by a yellow color, in a few drops of water, produced no effect with this test when diffused through six drachms. In operating with these tests, it is always advisable, therefore, to concentrate the liquid to the smallest possible bulk, and even to apply the tests to the dry residue.

There are no *objections* to this mode of testing, when we do not rely upon the action of sulphuretted hydrogen alone. The analyst must remember that some of the salts of mercury, silver, copper, cobalt, nickel, bismuth, iron, and tin, give a black precipitate with hydrosulphuret of ammonia; and, in some instances, with sulphuretted hydrogen. When, however, we obtain a white precipitate by diluted sulphuric acid, which is soluble in a solution of potash (first proved to be free from any trace of oxide of lead), and this alkaline solution is precipitated brown-black by sulphuretted hydrogen, the conclusion is that we are dealing with a salt of lead. Another portion precipitated by sulphuric acid will be found to be soluble in hydrochloric acid; and on evaporating this solution to dryness, and heating the residue with soda flux in the inner flame of the blow-pipe, we may procure a granule of metallic lead. Any of the compounds of lead thus treated, yield the metal, but the production of the metal is not absolutely necessary if the above results have been obtained. The iodide of potassium, and the chromate of potash, are useful as corroborative tests when applied to small dry residues. They both produce a rich yellow color.

Care must be taken that the potash used contains no lead. This alkali is generally contaminated with oxide of lead by reason of its being kept in a flint glass-bottle (post, p. 447).

The subacetate of lead, Goulard's water, and the other soluble salts of the metal, may be analyzed by a similar process. Goulard's water is a mixture of one drachm and a half of solution of subacetate of lead with a pint of water and a little spirit. The acids of the salts may be discovered by the tests elsewhere suggested. The nitrate of lead, when heated, yields red vapors of nitrous acid; the chloride fuses, and forms a fixed greenish-yellow mass; the carbonate yields an orange-colored residue of oxide of lead. Litharge may be examined by dissolving it in diluted nitric acid, and minium or red lead by digesting a portion of it in strong nitric acid. The *Carbonate* is dissolved with effervescence by acetic or diluted nitric acid: the *Sulphate* may be reduced by soda flux,—and the metal identified by its physical characters, or by the action of nitric acid, and the application of tests to the solution.

Lead in organic mixtures.—The acetate of lead is precipitated by many organic principles, especially by albumen and tannic acid. Thus, we may have to analyze either an organic liquid containing lead, or a solid precipitate consisting of mucus or mucous membrane, intimately united to the oxide of lead. The liquid must be filtered and examined by a trial test, *i. e.*, either by adding to a portion sulphuric acid, or by exposing bibulous paper dipped into the suspected liquid,—to a free current of sulphuretted hydrogen gas. If the paper be not stained brown, there is no perceptible quantity of lead dissolved;—should it be stained brown, we dilute the liquid if necessary, in order to destroy its viscidity, and pass into it a current of sulphuretted hydrogen gas until all action has ceased. The black sulphuret of lead should be collected on a filter, washed and dried, then boiled for a quarter of an hour in a mixture of one part of nitric acid diluted with four parts of water. This has the effect of transforming it, at least in part, to nitrate of lead soluble in water. This liquid may be either evaporated to dryness and the residue dissolved in water, or when filtered, it may be cautiously neutralized by potash (free from lead), or ammonia, and the tests added. If, on adding sulphuric acid, a white precipitate is formed soluble in pure potash, and the potash solution is turned black by hydrosulphuret of ammonia, there is sufficient evidence of the presence of lead. Should there be no lead dissolved, we may evaporate to dryness, carbonize by nitric acid, and redissolve the residue in water, or acid, as the case may require, for the purpose of testing.

Three portions of the nitric acid solution, obtained by either of these processes, may be evaporated to dryness on a glass slide, and the three tests,—sulphuric acid, iodide of potassium, and sulphuretted hydrogen, may be then applied to the dry residue.

When the quantity of organic matter is very large, it has been found advisable, in addition to carbonisation by nitric acid, to employ chlorate of potash, and to heat the mass (but not too violently) until all vapors have ceased to issue from it. The residue, finely powdered, may then be digested in nitric acid, in order to dissolve out any particles of lead.

Sometimes the carbonate or sulphate of lead (the latter as a result of antidotal treatment) are found in the form of a white powder in the stomach. They may be collected by decantation, and examined in the manner above pointed out.

Quantitative analysis.—This may be most conveniently effected, with respect to any of the soluble salts of lead, by passing into the solution a current of sulphuretted hydrogen gas, until the filtered liquid gives no longer any indication of the presence of lead. The precipitate should be well washed, dried, and weighed. Every 100 parts of sulphuret are equal to 158·3 of crystallized acetate: 138·3 of crystallised nitrate; 116·6 of chloride, and 111·6 of carbonate of lead. In some cases, it may be convenient to precipitate the lead by sulphuric acid, and to calculate the quantity from the sulphate,—washed and dried. 100 parts of this salt are equal to 26·4 of sulphuric acid, and 73·6 of oxide of lead.

Detection of lead in the tissues. Absorption and deposition.—The facts elsewhere recorded (ante, p. 66), render it unnecessary to discuss the question whether lead is absorbed and deposited in the tissues. Both in acute and chronic cases the metal lead, in some form, is found, more or less, in all the soft organs of the body. The blue line on the gums affords an instance of its deposition in this part of the body,—the color being probably due to the conversion of the deposited lead to the state of sulphuret. Lead was found by Tiedemann, in the blood of poisoned animals, and Prof. Cozzi, found it in the blood of a person laboring under lead-colic. Flandin did not succeed in detecting it in this liquid. The urine appears to be the great channel of elimination. Orfila found lead in this liquid, in the case of a woman who had swallowed an ounce of the acetate (Op. cit. i, 684), but Dr. Mitscherlich could not find it in the blood and urine of animals which he poisoned. In the case of a cow poisoned by lead paint, I found traces of it in the milk. Dr. Letheby states that, in his case of chronic poisoning (ante, p. 433), he detected lead in the brain, muscles, liver, intestines, blood, and in the serum found effused in the ventricles of the brain: but none was discovered in the bile or urine. Dr. Inman detected it in one case in the cerebellum (Med. Gaz. vol. xxxviii, p. 389; see also for its detection in the brain, Ed. Monthly Journal, July, 1851, p. 65). In a case already related (ante, p. 435), lead was discovered in the liver of a woman, and the symptoms thus received an explanation. It has been alleged that lead is a *normal* constituent of the body. In reference to this allegation, it may be remarked, that the lead hitherto found in the tissues is not normal but of an abnormal kind; and had the history of such cases been properly brought out, its introduction *ab extra* would have been demonstrated.

Although there are no facts to show that lead is a natural constituent of the body, yet the metal may be found in the tissues, in cases in which there can be no suspicion of criminal administration. Next to copper, no metal is so frequently met with in various articles of food as this (see post, p. 446); and as it is more slowly eliminated than other metals, it may accumulate in the tissues, and be occasionally discovered after death by chemical analysis. It is impossible, however, to raise a charge of poisoning on such a discovery. In November, 1843, a trial took place at the Assizes of the Puy de Dôme, in France, involving this question. The deceased died under suspicious circumstances: on an inspection of the body nothing was observed to indicate the action of an irritant poison, but the stomach was ulcerated, and in an otherwise diseased condition. No salt of lead was found in the contents, but *traces* of the metal were discovered on incinerating the viscera. The question then arose, whether the metal thus found was a natural constituent of the body, or the result of a portion which had been swallowed, and had acted as a poison. The medical opinions were conflicting. Orfila thought it was very probable, if not certain, that the deceased had died from the effects of lead (Annales d'Hygiène, Janvier, 1844); but the traces of lead were no doubt due to accidental causes,—they may have been taken in water, wine, snuff, or some other article of daily use.

The chemical processes above recommended for the detection of lead in organic liquids or solids, will be found equally serviceable for the tissues. In incinerating the dry tissue in a porcelain capsule, it will be advisable not to employ a higher temperature than is barely necessary for carbonizing and bringing the organic matter to dryness. Any particles of reduced lead may be separated from carbon and other substances by washing the residue in water, and decanting several times.

Lead has been so frequently detected in the soft organs and secretions, that the presence of it in the tissues of the body may now be looked for with some certainty, when in a case of poisoning it cannot be discovered either in the matter vomited or in the contents of the stomach after death. I believe that the liver, from its size and from the large quantity of blood it contains, is the organ best adapted for analysis. Here, as in the incineration of any of the soft parts of the body, the analyst is liable to be embarrassed by the presence of oxide of iron in the ash. This oxide may give a color with sulphuretted hydrogen gas, which, if this test alone were employed, might easily lead to a serious error. The sulphurets of lead and iron are, however, very differently affected by nitric acid; and on making the liquid rather strongly acid with this menstruum, we are quite sure that no sulphuret of iron will be formed. In an acid mixture of these two metals, brown sulphuret of lead only is precipitated. This precipitate, however, digested in strong nitric acid, is immediately converted into sulphate and nitrate of lead. In searching for the metal in the tissues, it is proper to remember that lead may be introduced accidentally into the ash by a crucible, or by other means which will easily suggest themselves; and as the tests for lead are of exceeding delicacy, it is the more necessary to use great caution in the steps of an analysis.

Drs. Wilson and Macadam infer from their experiments that the largest proportion of deposited lead will be found in the spleen (ante, p. 66), but this was in reference to a solitary case. They assign the following as the order of maximum deposit in the tissues,—the spleen, liver, lungs, kidney, heart, and coats of the intestines. Their process consisted in destroying the organic matter by boiling it until dissolved in nitro-muriatic acid. The acid liquid was diluted, filtered through calico, and evaporated to dryness. The dry residue was carbonized in a Hessian crucible—the ash digested in diluted nitric acid—evaporated and redissolved in hydrochloric acid. This acid solution was then precipitated by a current of sulphuretted hydrogen gas, and the precipitated sulphuret was converted to nitrate of lead by boiling it with diluted nitric acid. This was redissolved in water, and the usual indications of lead obtained by all the tests mentioned (ante, p. 442), including the chromate of potash. Hence, they recommend the selection of the spleen (Edin. Monthly Journ. Med. Sci. 1852, vol. xiv, pp. 386, 389).

The above case shows that lead may be easily detected in the tissues a fortnight after the taking of lead-poison has ceased; but M. L. Orfila has found lead in the tissues *eight months* after the withdrawal of the poison; and the facts connected with the slow disappearance of the blue line from the gums render it probable that traces of lead may be found after the lapse of one or two years. The reader will find some remarks on this subject ante (p. 58).

In reference to the detection of lead in organic solids—bread, cheese, snuff, &c.—it is commonly recommended to pour on a portion of the solid a solution of hydrosulphuret of ammonia; and if this be blackened, it is inferred that lead is present. This inference, however, without further experiments, would not be justifiable, since the presence of iron in the solid might give rise to a similar change. The only certain plan is to burn the organic substance, or to decompose it by heat, and to digest the carbonaceous ash in nitric acid, slightly diluted. The acid liquid should be filtered, and tested by the appropriate tests.

LEAD AS AN ACCIDENTAL INGREDIENT IN ARTICLES OF FOOD OR DAILY USE.—Liquids used for culinary or dietetic purposes, especially if they contain a free acid, are liable to become impregnated with oxide of lead, derived from the *glaze* of the vessel in which they are kept, and thus to form poisonous salts. If vinegar be used, acetate of lead may result. Litharge-glaze is easily dissolved by alkaline or fatty substances. The eating of dripping, or the fat of meat, baked in a newly-glazed vessel, has thus been known to give rise to a slight attack of colic, while the symptoms were referred to some substance mixed with the food. A case in which the whole of the members of a family were thus poisoned, will be found in the Lancet (July 4, 1846, p. 27). Another instance of a similar kind is reported in the Medical Gazette (vol. xlvii, p. 659). All newly glazed vessels yield a larger or smaller proportion of lead on boiling them in pure acetic acid, or a solution of potash free from lead. In this manner the poisonous nature of the glaze may be tested: the oxide of lead being dissolved by the acid or the alkali. I have found common acetic acid itself to contain, as impurity, two per cent. of acetate of lead.

Lead may be an accidental ingredient in distilled water, in the waters of the essential oils, as well as in certain medicines. Some of these acquire an impregnation of it in the process of manufacture—*e. g.* carbonate of ammonia, which is sublimed into leaden vessels; carbonate of soda, borax, and other salts, when crystallized in leaden pans. M. Chevallier has lately pointed out a contamination of tartaric acid with lead, in the proportion of 1·2 to 1·5 per cent. He believes that this arises from the employment of lead to sink the strings in the crystallising solutions. (Journal de Chimie, Juin, 1858, p. 354.) Solutions of alkaline salts kept in flint-glass bottles are generally contaminated with lead. The alkalies, potash and soda, their carbonates and bicarbonates, the alkaline silicates, phosphate of soda, and some others, are thus rendered impure and unfit for chemical use; but the quantity of lead present is not sufficient to produce symptoms of poisoning.

I am indebted to Mr. Procter, of York, for the particulars of a case of some novelty, in reference to the contamination of food with lead. In July, 1852, four men partook of rhubarb-pie and *milk* for supper. Shortly afterwards, they were all seized with violent vomiting and intense colic. A portion of the vomited matters and food was examined by Mr. Procter, and lead was detected in them. The only source to which the lead could be traced, was the glaze of the pans in which the milk was kept. Lead pipes are largely used by publicans for the supply of *beer*. It is possible, therefore, if the beer is acid, and allowed to remain some time in the pipe, that it may acquire an impregnation of lead, which might give rise to colic, and other unpleasant symptoms, creating a suspicion of criminal poisoning. *Cider*, which is a highly acid liquid, is apt to become poisoned with the salts of lead when it comes in contact with this metal. It has been generally supposed that the only poisonous compound produced in this case is the insoluble malate; and it appears from an accident which occurred in France, whereby six persons were seized with symptoms of lead-poisoning from drinking cider, that Chevallier and Ollivier discovered that the salt which caused the symptoms was the malate of lead. A large quantity of acid may probably dissolve this and other vegetable salts which are reputed to be insoluble; or, like the carbonate of lead in water, the insoluble salts may be diffused through the liquid, and thus taken in an extreme state of division. In some instances, the carbonate of lead itself may be formed and act as the poison. A case of this kind has been already given. (See ante, p. .432) An instance of chronic poisoning occurred in an American family, by reason of the members of it drinking cider which had been poured into vessels newly painted with white lead and linseed oil. No effect was observed for a fortnight. The chief peculiarity in the symptoms was a soreness in the soles of the feet, succeeded by slight nausea and a vesicular eruption. (Amer. Jour. Med. Sci. July, 1843; and Trans. Prov. Assoc. vol. i, p. 119.) In another set of cases, colic and constipation followed the use of cider which had acquired an impregnation of lead from its having been passed through a leaden funnel. Lead was detected in the cider in the proportion of 1-4000th part, or about seventeen grains in the gallon. The urine of one of these persons was found to contain lead. (L'Union Médicale, February 17, 1857; and British and Foreign Medical Review, vol. xix, 1857, p. 499.) When liquids of this kind are impregnated with oxide of lead, the fact is immediately known by their being turned more or less of a brown colour by hydrosulphuret of ammonia.

Pork is frequently salted in leaden vessels, and is allowed to remain in such vessels soaking in the brine. The effect of this is to impregnate the pork with a portion of chloride of lead,—its color and taste have been observed to be affected under these circumstances. Litharge was formerly much used to remove the acidity of sour *wine*, and convey a sweet taste. Acetate of lead, or some other vegetable salt of the metal, is in these cases formed; and the use of such wine may be productive of alarming symptoms. Many years since a fatal epidemic colic prevailed in Paris owing to this cause;—the adulteration was discovered by Fourcroy, and it was immediately suppressed. Such wine is known by its being blackened by hydrosulphuret of ammonia. Lead-shot are much employed for the purpose of cleaning wine-bottles, and pellets are frequently left in the bottles. A question has arisen, whether wine introduced into them is liable to acquire a poisonous impregnation from lead. I have found, when the shot are in much larger proportion than could ever be left by accident in a wine-bottle, that good wine, whether port or sherry, is only slowly impregnated with lead. After two or three months a white sediment had formed, but no lead was dissolved; after thirteen months the port wine retained its color, and scarcely any portion of lead was dissolved in it: the sherry had become darker in color, and the presence of lead was very evident in it. The undissolved salt of lead in the sediment will, however, if swallowed, produce all the effects of chronic poisoning. Very acid wines (from acetic acid), such as those made from the currant or gooseberry, may, however, be rapidly impregnated with the metal, and in a quantity sufficient to produce colic or other serious symptoms. *New rum*, as it is made in the West Indies, often contains lead derived from the worm of the still, and lead-colic frequently attacks those who drink it. *Old rum*, on the other hand, is by no means unwholesome, and is therefore in great demand. Dr. Traill gives the following explanation of this difference in properties. He found that the rum which was received in glass bottles from the still, was always impregnated with lead; but when kept in oak casks, the tannin of the oak is slowly dissolved by the spirit, and precipitates the lead in an insoluble form, the spirit thereby becoming perfectly wholesome. He has suggested that a little decoction of oak bark, added to the new rum, would render it equally innoxious. (Outlines, 112.) Mr. Scanlan has called attention to the fact, that oxide of lead is sometimes present in *distilled water*, when leaden pipes have been used for the purpose of condensing the vapor. It appears, however, to be rapidly converted to carbonate, and thus rendered insoluble. (Pharm. Journ. Aug. 1844, p. 69; also Dec. 1845, p. 279.)

Flour.—In 1857, several families at La Tremblaie, in France, suffered from symptoms of lead poisoning. On analysis, a salt of lead was found in large quantity in the flour and in the bread. An inquiry into the facts led to the discovery that part of the grinding machinery of the mill had been stopped with lead cement, and this was covered with plaster. The plaster had given way, and the salt of lead which fell out was ground and mixed with the flour. (Journal de Chimie, 1857, p. 278.)

Sugar.—A few years since, an attempt was made in this country to work a process for the refining of sugar by the use of subacetate of lead, the surplus lead being afterwards precipitated in the syrup by a current of sulphurous acid. The late Dr. Pereira, Dr. Carpenter, and myself, were required by Government to report on this process in reference to its probable effect on public health. We found that the lead was not entirely removed from the refined white sugar, but that a variable quantity remained in it under the form of sulphite of lead; and there was good reason to believe that a still larger proportion was carried into the treacle. Our report was decidedly adverse to the project. It has been found that sugar, as it is ordinarily manufactured, is sometimes the medium of conveying lead-poison into the system, and giving rise to attacks of colic in those who partake of it. Dr. Jackson has reported an instance of this kind, in which several persons lost their lives, and many others were attacked with paralysis and colic, who had partaken of sugar which had probably been kept in leaden reservoirs. Lead was discovered in the sugar in large quantity. (Med. Gaz. xvii, 1036. See also Beck's Med. Jur. vol. ii, p. 646.) Sugar, refined by the ordinary process, may contain traces of lead. The metal cones into which the syrup is poured are painted with white lead; and this requires occasional renewal,—a proof that the loaf of sugar must be more or less contaminated.

Snuff.—Some varieties of snuff are adulterated with lead to a degree to cause symptoms of chronic poisoning. The compounds of lead used for this purpose are the red oxide (minium), and the chromate of lead; the object of the adulterator being to improve the color and the saleable value of the snuff, at the expense of the health, and it may be of the life, of his customer! Out of forty-three samples of popular kinds of snuff examined by Dr. Hassall, chromate of lead was detected in nine, and oxide of lead in three samples. The chromate varied in quantity from one to four and a half per cent., and the oxide (red lead) reached as much as three per cent.! (Food and its Adulterations, p. 591.) From this statement, it will not be surprising that snuff should occasionally cause symptoms of lead-colic, or even death. (Med. Gaz. vol. xxxii, p. 138; also Ann. d'Hyg. 1831, vol. ii, p. 197.) In a case in which I was consulted a few years since, I have reason to believe that snuff, adulterated with lead, led to a series of constitutional symptoms which ultimately destroyed the life of a gentleman. This subject has lately been investigated by Dr. Meyer, of Berlin, and the results are most unsatisfactory for the takers of snuff. 1. A man, æt. 38, was seized without any apparent cause, with paralysis of the extensor muscles of the three middle fingers of each hand. In two months there was a considerable projection of the wrists. It was discovered that the snuff which he had been in the habit of taking contained a large proportion of lead. The use of this snuff was discontinued, and the paralysis disappeared under treatment. 2. A man, æt. 43, used snuff from the same factory. He suffered from disturbed digestion and colic. In February, 1855, he was attacked with paralysis, involving first the fingers, and slowly extending to the muscles of the forearm and shoulders. There was loss of sensation and motion in the extensor muscles, and yellowness of skin. This man recovered in a year. 3. A similar case. There were colics, with paralysis of the arms and hands in 1852, and of the legs in 1854. There was also wasting of the extensor muscles. A discontinuance of the snuff led to the disappearance of the colics, and improved the condition of the patient. 4. A physician, æt. 45, in the habit of taking snuff. There was complete paralysis in the upper limbs in 1851, and of the lower limbs in 1854. The cause was not suspected; but when discovered and removed, there was a rapid cure. 5. A man, æt. 50. There was paralysis of the fingers following the use of snuff, which ceased on a removal of the cause. (Journal de Chimie, Juillet, 1858, p. 394.) Dr. Meyer regards the following as the most prominent symptoms: paralysis, affecting chiefly the extensor muscles of the arms; yellowness of the skin; prominence of the metacarpal bones; colics and weakness, with wasting of the extensor mucles of the hands. The great danger in these cases is,—that the real cause of the symptoms may escape notice until they are too far advanced for cure.

Apart from wilful adulteration, snuff and tobacco are liable to acquire an impregnation of lead from being kept in vessels or wrappers made of lead, or of a spurious alloy of lead, called "patent tin-foil." In a Prussian police ordinance for May, 1857, the public are warned of the danger of purchasing snuff packed in leaden wrappers. It is there stated that several cases of lead-paralysis have occurred from the use of this snuff, as it is, under these circumstances, frequently impregnated with lead. (Casper's Vierteljahrschrift, Jan. 1858, pp. 184 and 163.) *Chocolate* is also sold in wrappers of this kind; and the chromate of lead is sometimes used for the purpose of coloring confectionery sold to children. An important case was tried at the Guildhall Summer Sittings, 1857 (Adnam v. Betts), which, for the first time, revealed an extensive source of lead-poisoning for the infant population of this country. The plaintiff, who was a manufacturer of groats as an article of food for children and invalids, claimed damages of the defendant on the ground that the food had been damaged, and rendered noxious and unsaleable, by reason of its having been wrapped in a spurious metal, sold to the plaintiff as tin-foil. Mr. Brande, Mr. Scanlan, and myself examined the packets of food; and we found that the metal wrappers were extensively corroded in a number of small holes, and the inside layer of the food was discolored, and strongly impregnated with lead. On examining the metallic wrapper, sold as Bett's patent metal, or tin-foil, we found it to consist of from seventy to eighty parts of lead, and of twenty to thirty parts of tin! The tin gave merely a facing to the lead, and made it appear like tin-foil. The plaintiff lost his case chiefly on the ground of his having purchased the metal at a price at which he ought to have known pure tin-foil could not be sold. Assuming this to be a good answer in law, it is fair to question the propriety of a patent being legally granted for the sale of such a spurious and noxious alloy as this. It is sold for wrappers and capsules, and is placed in contact with liquids and solids used as articles of food; it is therefore liable to impart to them a dangerous impregnation of lead. If a man sells copper, faced with gold, as patent gold, he may be punished for fraud, although the damage is here only of a pecuniary nature. The sale of *lead-foil* as tin, thus legally licensed, may, by its use, lead to extensive injury of the health of a population. An alloy of lead and tin was employed, a few years since, in the form of a screw-capsule, as a patented substitute for corks in bottling wines, preserves, &c. On examining the preserved fruits, and the vinous liquids kept in bottles thus stopped by patented noxious stoppers, I found them to be strongly impregnated with lead! In France, the sale of this article as tin or tin-foil carries with it certain penalties. In a recent case, proceedings were taken by a purchaser against the vendor. It was proved that the so-called tin-foil contained a large quantity of lead; and the vendor was summarily condemned to a month's imprisonment, 150 francs fine, and costs, as well as a return of the money paid for the metal, which was ordered to be confiscated by the court. (Journal de Chimie, Jan. 1858, p. 50.) Many of the *aërated water* vessels now sold are provided with stoppers consisting of an alloy of lead. In some of these an incrustation of carbonate is produced, which is liable to be detached and taken with the water. I have heard of a case in which the symptoms of chronic lead-poisoning resulted from the use of such water. *Hair-dyes* and *cosmetic powders* are notoriously compounded of lead! I have elsewhere referred to cases of paralysis produced by the use of such substances (ante, p. 434).

Water.—Of all articles of diet, there is none which has been so fruitful a source of lead-poisoning as water. The symptoms and appearances have been elsewhere described (ante, p. 436). It will now only be necessary to consider the circumstances under which water, distributed in leaden pipes, or stored in leaden cisterns, may acquire a poisonous impregnation. 1. Absolutely pure water, recently boiled to deprive it of air, and placed in contact with polished lead in a hermetically-sealed tube, has no chemical action on the metal. 2. The same water, exposed to air, produces in a few minutes around the surface of the metal a milky-looking film; and, in twenty-four hours, this shows itself as a white compound, diffused in pearly scales either loosely adhering to the lead, or as a white sediment at the bottom of the vessel. The compound thus formed is a mixture of hydrated oxide and carbonate of lead, the carbonic acid and oxygen being derived from the air. This compound is not dissolved in the water to any perceptible extent, but is mechanically diffused through it. Water in this state is, however, just as dangerous to health as if the lead wère dissolved. The more nearly pure, or the more free from saline matter the water is, the more intense is this chemical action; and it commonly proceeds until all the lead is converted to this chemical compound, or until the surface of the metal is invested with so closely adhering a film, that neither oxygen nor carbonic acid can reach it. Water thus contaminated, if passed through a good sand and charcoal filter, will lose the whole of the salt of lead, and be rendered innocuous. 3. *Rain*, *snow*, and *ice water* (Wenham ice), being generally remarkably free from saline matter, act in a similar manner upon lead. An epidemic of lead-colic which appeared many years since, at Amsterdam, was traced to the use of rain-water collected from leaden roofs. (Christison on Poisons, p. 526.) Rain-water which has passed over slate or tile does not, however, readily act upon lead. 4. *Pure*, or *soft spring*, or *lake* water, containing only a few grains of saline matter to the imperial gallon, has been hitherto considered as dangerous for use. The water supplied to Tunbridge, in 1815, through leaden pipes, was what is called a pure water, and its use caused an outbreak of lead-colic in that town. The water of Claremont, containing only five grains of saline matter to the imperial gallon, produced, in a few months, a severe form of lead-colic (ante, p. 437) in the Royal family of France, elsewhere described. The water of the Surrey sands has an evil reputation in this respect: it is comparatively pure, and, generally speaking, acts strongly on lead. The severe cases of lead-colic met with by Dr. Thomson (ante, p. 436) were traced by him to the Surrey-sands' water. The chief part of the saline matter is common salt. The water supplied through leaden pipes to the Royal kennels, at Ascot, caused, a few years since, a general lameness (from lead-paralysis) among the hounds. In that neighborhood I have found one sample of water to act powerfully on lead, and another not, although both would come under the head of pure waters.

In the year 1854, the influence of *Lake water* on lead underwent a close scrutiny before a committee of the House of Commons, in reference to the proposed supply of Glasgow with the pure water of Loch Katrine, containing only two grains of saline matter to the imperial gallon. I found this water to act strongly on lead when the bright and highly polished metal was immersed in it under a free exposure to air. It had no action on lead when the metal was in its ordinarily dull state. When this water was allowed to stand some time over masses of limestone and old red sandstone, its chemical action on lead ceased. The question of the safety of supplying such a water as this through leaden pipes to a large and populous city, gave rise to a great difference of opinion among a large body of scientific chemists, who were examined before the committee. The question, however, was finally decided by an appeal to the experience of other towns in which a water of similar quality had been many years in use. Inverness had been supplied from Loch Ness, through leaden pipes, with water as pure as that of Loch Katrine; and during a period of twenty-four years there had been no cases of lead-colic in that town from the use of this water. A remarkable fact transpired with respect to the Ness water. A portion drawn from a leaden pipe in a private house, at Inverness, contained no lead; but when the water was placed in contact with a bright surface of the metal, it rapidly acted on it. The town of Whitehaven, which is supplied from Enerdale Lake, presented another instance of the distribution of pure lake water through lead, without any accident occurring from its use, in a large population. Yet the water of this lake exerts a chemical action on bright lead. On these practical grounds and proved results, Glasgow was permitted to receive its supply from Loch Katrine.

There is no doubt that, in these cases of non-contamination, the leaden pipe or cistern soon acquires a closely adhering deposit, which is sufficiently thick to prevent any further chemical action of the water on the lead beneath. The facts above mentioned show, that if the common mode of testing water, by immersing in it a bright and highly-polished surface of lead, were to be taken as a criterion of danger or safety, the towns of Inverness and Whitehaven should be immediately deprived of that supply of good water which they have now enjoyed, with perfect immunity, for more than sixteen years! We also learn from them, that whatever scientific theories may exist regarding the poisoning of water by lead,—the question cannot be determined from the known constitution of the water; it should be always based on actual experiment. The lead should be used in the state in which it is ordinarily employed for cisterns by the plumber (six pound lead). A portion of the lead, exposing about sixteen or twenty square inches, should be immersed in twenty ounces of water, so that part of the metal is out of the water. The chemical change, if any, should be daily noticed, and the water tested by a current of sulphuretted hydrogen gas in a glass tube, about twelve inches long and one inch in diameter. By looking down the length of the tube, after the gas has been passed into it for a sufficient time to impart a smell, the slightest change to a brown color will be immediately perceptible. The quantity present in a given sample may be estimated by passing the gas into similar tubes containing minute but decreasing fractional proportions of a grain of lead in a gallon of water. A comparison of the shade of color with the shades of the standard tubes, will thus enable the operator to fix the proportion of lead with sufficient accuracy for practical purposes.

It is necessary to bear in mind, that a water may contain a noxious proportion of lead, but present no opacity: the lead may be *dissolved* in it as chloride or nitrate. In this case it may be tested in the entire state; and if this should fail to reveal the presence of lead, half a gallon of the water may be evaporated, and, towards the end of the operation, the residue may be dissolved in diluted nitric acid, and then tested. The quantity of lead present in water which is noxious to health, is usually very small. In the Claremont case, Dr. Hofmann found only one grain of lead in a gallon; but even one-fourth of this quantity would, in time, be productive of serious injury to health (ante, p. 437; and Med. Gazette, vol. xliv, p. 261).

The quality of the *lead* appears to exert some influence on the results. Certain kinds of lead are speedily acted on and corroded by water; others with difficulty. In the experiments of Loch Katrine water, Dr. Miller and I found that the lead supplied for our small cisterns obtained from different parts of London, when used in the state in which it was received, produced no chemical changes with the water; whereas Dr. Penny found that the lead supplied to him for a similar purpose, in Glasgow, was rapidly attacked and corroded. The cause of these differences is not at all understood; but one fact is certainly established,—the more highly polished and the brighter the lead, the greater is the probability that, *cæteris paribus*, water will be rendered noxious by contact with it under exposure to air. An alloy of lead with tin retards, but does not prevent chemical action.

5. *River water. Hard spring water.*—River and spring waters, containing a moderate amount of saline matter (twenty to forty grains in the gallon), do not, in general, give rise to a noxious chemical action on lead, provided they contain sulphate and carbonate of lime, and not too

large a proportion of alkaline chlorides or nitrates. The Thames River water, containing about seventeen grains of saline matter in the gallon, is remarkably free from this action; and when it is considered that it is supplied through lead, to the extent of 80,000,000 of gallons per diem, to a population of nearly 2,500,000, and that cases of lead-colic from the use of London water are unheard of,—it is obvious that it is so constituted as to prevent these dangerous chemical changes. The principal salts in this water are carbonate and sulphate of lime, with a small proportion of common salt. Thames' water frequently contains alkaline nitrates, and these salts may be the means of giving to it, occasionally, an impregnation of lead. The alkaline chlorides also favor the formation of chloride of lead, which the water may hold dissolved; and, at the same time, they do not prevent the production of the noxious carbonate. Chloride of sodium appears to be the chief ingredient in the Surrey-sands' water. The Claremont water contained five grains of saline matter in the gallon, of which one-half was common salt. In the Surrey-sands' water, which caused the symptoms in Dr. Thomson's cases, the quantity of mineral matter amounted to about five grains and a half in a gallon, and four-fifths of this were common salt. Some kinds of spring-water, containing a large proportion of sulphate of lime and other salts, are found to act powerfully on lead. Mr. Osborne considers that lead, in the form of chloride, is liable to be produced in the spring water of the wells around Southampton. (Pharm. Times, Sept. 26, 1846, p. 64.) Artesian water, by its alkaline salts and the large proportion of neutral salts contained in it, is found to act upon lead. In short, both hard and soft water may be rendered noxious by contact with lead, according to circumstances but little understood. All kinds of theories have been put forward to account for these chemical changes; but none are satisfactory, and none will explain all the facts. An excess of saline matter in some cases, and a total deficiency of it in other cases, may equally produce noxious effects. Sulphate and carbonate of lime,—which, in small proportion, appear to be beneficial,—are injurious when in large quantity; and the alkaline nitrates and chlorides appear to be injurious under all circumstances. It is a curious fact, that lead may be kept immersed in a sample of water, unchanged; while the vapor of that water, as it rises (distilled water), will corrode a leaden cover placed over the vessel, and thus impregnate the water below. As a general rule, soft and pure waters act upon lead, and hard waters do not; but a water must not be condemned as dangerous because it is soft, nor approved as safe because it is hard. In reference to some soft waters, the chemical action soon ceases; while in others it appears to continue unchanged so long as there is any metal under free exposure to air; but this rule is not so fixed as to dispense with the necessity of a special examination in each case (ante, p. 453). The variable intermixture and proportion of salts and gases contained in natural waters render it impossible to state, *à priori*, what will be the results of experiment.

When a water continues to act powerfully on lead and iron, and there are no means of changing the supply, it would be proper to substitute well-tinned pipe, glass, or earthenware, for lead. A cistern of slate should also be substituted for one of lead. For drinking purposes, the water should always be filtered through a bed of sand. These rules should be adopted when there is a tendency to act upon lead, whether the water be soft or hard.

POISONING OF CATTLE BY LEAD.—Medical evidence has of late years been required in cases in which damages have been claimed for alleged loss of cattle by reason of the vicinity of lead-works. I have elsewhere referred to two cases (ante, p. 66),—one in which a mare died from drinking at a stream into which carbonate of lead was discharged from certain lead-works. Dr. Wilson conducted the examination in this case: the cause of death was clearly traced to lead, and a quantity of this metal was separated from the organs. Lead was found in the water of the stream, and in the vegetables growing on the soil. (Ed. Monthly Journal, 1852, vol. xiv, p. 386.) In the case of Stephens v. Barwell (Wells Aut. Assizes, 1855), it was alleged on the part of the plaintiff that a large number of sheep and cattle had been destroyed by fumes of lead escaping from a chimney on the defendant's works. The case involved this curious point,—namely, admitting the sheep and cattle to have been destroyed by lead (of which there was not much doubt, at least in some instances), whether the lead was deposited on the herbage from the defendant's chimney, or taken up by the plants from the soil, and incorporated with their tissues. Mr. Brande and I were required to examine the defendant's flue, and found, from its enormous length (upwards of 2000 feet), and the constant cooling and washing of the vapors which traversed it with a large quantity of water, that every reasonable precaution had been taken to prevent their escape. In going over the plaintiff's ground, we could not perceive on the herbage, far or near, the slightest appearance of a deposit of white lead, or of lead in any form. From the local position of the chimney, and the contiguous hills of the Mendip, it also appeared quite impossible that the heavy vapors, if any, of carbonate of lead, could have been deposited so as to have destroyed the cattle at one spot where some were said to have pastured. It turned out, on further inquiry, that at five or six miles' distance, and in various directions across the Mendip, quite remote from the defendant's chimney, animals had been known to die occasionally from lead-poisoning; or, to use the local term, from being "moindered." We also found that sheep and cattle had died from lead-poisoning on and around the spot then complained of, before the defendant's works had been erected; and that there was no relation, either in time or distance, between the working of the lead-ore and the deaths of these cattle. This led us to examine the soil, herbage, and water of the plaintiff's fields in which the animals were stated to have been poisoned. The result was, that the soil of some of the fields in which the cattle had pastured consisted of the disintegrated slag of ancient lead-works loosely mixed with surface mould; the proportion of lead, as mixed with silicate and carbonate of lead, varied from 1·6 to 2·4 per cent. The herbage of all kinds growing in these fields, consisting of grass, weeds, furze, thistles, and shrubs, although their growth was unaffected, yielded a quantity of lead in their ashes. The examination of the water of a pond and brook from which the plaintiff's cattle drank, showed that, although the water itself contained no lead dissolved, the fine sedimentary matter mechanically diffused through it consisted of silicate of lead with a small proportion of carbonate and a small quantity of arsenic. The fine dust, or sediment of a drain leading directly to this cattle-pond yielded on analysis 0·43 grains per cent. of metallic lead. The ancient slag with which the roads about the plaintiff's farm were repaired contained 9 per cent. of lead and a quantity of arsenic, estimated at from half a grain to a grain in the pound. The whole drainage of these roads passed into a pond used by the plaintiff for watering his cattle.

Considering that it was a novel and important question to determine whether plants could take up lead from a lead soil and thus render herbage poisonous, we brought to London a quantity of earth taken from four of the plaintiff's fields, in which the greater number of his sheep and cattle were reported to have died. Mustard and cress seeds were sown in these four samples of the Mendip leaden soil; and, for a comparative experiment, in a sample of ordinary garden-mould. In about eight days, the crops were carefully cut, without interfering with the earth, and submitted by Mr. Brande and myself to a chemical examination. We first satisfied ourselves that there was no lead on the *outside* of the plants by washing them in pure diluted acetic acid and testing the washings. The vegetable matter was then dried and burnt, and lead in well-marked quantity was found in the ashes. The plants grown on the London mould did not yield lead either on the outside or inside. Samples of grass of the plaintiff's fields on which the animals pastured were now examined by a lens. There were no stains of white lead, such as would be deposited from a flue, but a brownish-colored, loosely adhering powder, easily brushed off, and obviously a part of the dust of the soil in which the grass grew. The acetic acid washings of this grass yielded lead; and after the whole of the lead had been thus removed from the surface, a quantity of the grass was dried and burnt. The ashes gave a residue of lead in notable proportion. One sample of grass from a field belonging to the plaintiff, gathered and given by himself to the defendant, yielded lead in the ashes only; there was no lead obtained from the washings of the outside. It was thus conclusively proved: 1. That plants growing on this leaden soil took up lead into their tissues, and that a portion of the lead found in the herbage of the plaintiff's fields had been thus imbibed from the soil. (See paper by Dr. Wilson, Ed. Monthly Journal, 1852, vol. xiv, p. 386.) 2. That the outside of the herbage presented no appearance of white deposit of carbonate of lead, but merely the brown dust of the leaden soil. 3. That the sedimentary matter in the pond and brook from which the cattle drank, consisted of the finely powdered lead-slag of the district, combined with traces of arsenic.

The evidence given at the trial showed that sheep and cattle belonging to plaintiff had died from lead-poison; and Mr. Herapath was called to prove that the sole source of that lead-poison was the flue of the defendant's works. In answer to questions in cross-examination, he stated, however, that he had not analyzed the earth on the plaintiff's grounds, as that was no part of his case. He had analyzed the lead-slag of the district, but had not looked for arsenic in it. He had never found lead in the ashes of plants, but he had never examined for lead the ashes of plants growing on a leaden soil. Although he had made no experiments on the subject, he did not believe that growing plants would take up lead from the soil and deposit this metal in their tissues. As a chemist, he undertook to say it was impossible to determine whether lead was in the substance as well as on the surface of a plant. In this case, although he had made no trial to detect it in the tissues of the plants, he believed that it was entirely upon the surface, and deposited on them exclusively from the defendant's works. The plaintiff's pond and brook water contained no lead in a *dissolved* form: but he had not examined the sediment, although the animals might drink the water in a turbid state!

As a proof that white lead from the flue was deposited on the surrounding vegetables, a branch of a tree was produced in court, the whole surface of which presented numerous white spots or stains, such as might have resulted from dipping it into the washings of white lead in water. That this had been the mode in which the stains were produced was rendered highly probable among other matters, by the curious fact, that the cut surface of the branch presented in the fresh wood similar white stains! This branch was very judiciously not produced in the evidence, although brought into court apparently for that purpose.

The case was met on the part of the defendant by the statement, that cattle had died from lead-poisoning on this farm before the works were in operation; that they had died from the same cause in spots remote from these works, wherever the pasture of the district happened to be over lead-slag, so that some localities had acquired an evil name from this circumstance, and the pasturing of cattle in such places had been avoided: that there was a sufficient cause of lead-poisoning in the herbage grown on the soil, which abounded in lead, and in the water which the cattle drank; and there was a total absence of proof of any deposit of lead on the surrounding vegetation. Upon this statement, and without any evidence being called for, an arrangement was made between the parties.

This case shows that a charge of poisoning cattle may be plausibly made, and even apparently sustained by pseudo-scientific evidence, when a proper examination of the facts may lead to the conclusion that the charge is wholly unfounded or unproved. As an "expert," and well acquainted with this locality, Mr. Herapath might have analyzed the soil of the fields, and have tested by experiment the question,—whether growing plants would or would not imbibe lead from the earth, instead of denying a fact on which he had had no experience. Such an analysis was in every respect necessary, not merely for the sake of public justice, but, if the claim for damages were well-founded, for the interest of the client who retained him. In his evidence he stated that he had analyzed the slag of the district, and he found it to contain 36 per cent. of carbonate of lead, the compound which he charged the defendant with diffusing over the plaintiff's grounds. The same slag was plainly visible in the plaintiff's fields, and the result of an analysis might have been unfortunate for his client's claim. Supposing, however, that he had analyzed the mould of the field, and found no lead, it would have been a strong point in favor of his client. He also stated in his evidence, that he had examined the waters of the plaintiff's pond and brook, and found no lead *dissolved* in them; but he did not examine the sediment, although he very well knew that cattle do not drink filtered water, and that a fine lead sediment diffused through water may be just as poisonous to cattle as lead in a state of solution! The result of this analysis would have probably been highly inconvenient to the plaintiff's interests. In reference to the examination of three or four dead animals, he stated that he had found no lead in the liver! He believed they were poisoned in dry weather by drawing the lead-dust through their nostrils into the lungs while pasturing, and in wet weather the lead would pass with the food into the stomach! He did not, however, find any powder or dust in the lungs. He confined his analysis to an examination of the *outside* of the plants only: he did not examine the tissues of the grass, as acids would act upon and destroy them! He believed that the lead was on the outside of the grass, and had been there deposited from the defendant's flue.

Such was the evidence upon which it was attempted to base the plaintiff's claim. It will be perceived that the case admirably admitted of that sophistical application of scientific facts which is sometimes apparent in a bad or weak cause. Two propositions are laid before a jury, —one is proved, the other not,—and the jury are invited to accept proof of one as proof of both! The cattle had died of lead-poison, but it was studiously kept from the knowledge of the jury that the plaintiff had a store of lead-poison on his premises, which would account for their deaths. This case might suggest many comments regarding the reception of scientific evidence when made a matter of business; but all will agree that this plan of carrying a chemical analysis only so far as it may serve the cause of a plaintiff or defendant, is most damaging to the cause of science and to the credit of scientific witnesses.

Sometimes this form of poisoning is purely accidental. Dr. Gordon, of Aberdeen, lost seven head of fine cattle, which were grazing in a field. On opening the stomachs, white lead was found therein. The deaths were here traced to some refuse paint or white lead, which had been carelessly left about as waste. The sudden deaths of animals may be frequently due to a cause of this nature, and suspicion fall unjustly upon parties who have the care of them. Poisonous materials of this kind should be effectually disposed of, and not thrown into ash-pits or on waste land.

CHAPTER XXX.

COPPER—EFFECTS PRODUCED BY THE METAL AND ITS ALLOYS. BLUE VITRIOL—VERDIGRIS AND OTHER SALTS—SYMPTOMS—CHRONIC POISONING—EFFECTS OF EXTERNAL APPLICATION—APPEARANCES AFTER DEATH—FATAL DOSE—TREATMENT—CHEMICAL ANALYSIS—TESTS. COPPER IN ORGANIC LIQUIDS—IN THE TISSUES—IN THE SOIL OF CEMETERIES—IN ARTICLES OF FOOD.

General Remarks.—Copper itself is said to be destitute of poisonous properties; but it would appear that when alloyed with other metals and reduced to a finely pulverulent state, it may act as a poison. A singular instance of this kind is on record. The printing in gold, as it is termed, is performed by means of a species of brass or copper alloy. The letters are printed with a mixture of size and gamboge; and the copper alloy, reduced to such a fine state of division that it floats in the atmosphere in an impalpable dust, is then brushed over the surface. A boy employed in this occupation was, on the third day, seized with vomiting of a green-colored fluid, heat and constriction of the gullet, pain in the stomach, loss of appetite and rest, and a severe itching in all those parts which were covered with hair. These on examination were found to be of a deep green color. The boy soon recovered. About twelve other persons, employed in the same work, suffered from similar symptoms; but this did not prevent them from continuing the work. The poison in this case probably entered the system through the lungs and skin. This peculiar effect of finely divided copper in giving a green tint to those parts covered with hair is mentioned by Dr. Falconer in his Essay on the Poison of Copper (p. 42), published in 1774.

An alloy of copper, made to resemble gold, is largely used in the ornamenting of gingerbread and confectionery. I am not aware of any accident having occurred from its being thus eaten; but it is a noxious practice, and in France is especially prohibited, under a penalty, by police regulations. (Journal de Chimie, Février, 1847.) This alloy is easily known from gold by its entire solubility in nitric acid, with which it forms a greenish-colored solution of nitrate of copper. When metallic copper is swallowed, colicky pains and other symptoms sometimes follow in consequence of the metal becoming partially oxidized and dissolved. The experimental researches of M. Leportier show that the pure metal is not poisonous (Ann. d'Hyg. 1840, ii, 99); but it may cause death as a mechanical irritant. (See ante, p. 25.)

Copper coins, when swallowed, may produce a certain amount of poisonous salt from the action of the alkaline chlorides in the stomach; but the great danger to be apprehended in these cases is that they are liable to cause death by a mechanical action. (See case, ante, p. 29.)

All the salts of copper are poisonous. The two most commonly known are the *sulphate, blue vitriol, blue stone*, and the *subacetate* or *verdigris*. These substances have been frequently taken and administered in large doses for the purpose of suicide and in attempts at abortion and murder. In the latter case the attempt has been immediately discovered, owing to the strong metallic taste as well as color possessed by the salts. The taste would in general render it impossible that a poisonous dose of blue vitriol or verdigris should be taken unknowingly. With the exception of the wilful use of these salts, poisoning by copper is commonly the accidental result of the use of this metal for culinary purposes.

SYMPTOMS. ACUTE POISONING.—Poisoning by copper may be divided into the acute and chronic forms. Cases of acute poisoning from blue vitriol or verdigris are occasionally met with;—but the symptoms have nearly the same character and course in reference to these and all the other compounds of copper, if we except the arsenite, which has been already considered among the arsenical poisons (ante, p. 385). When the sulphate is taken in doses of half an ounce or upwards a strong metallic taste is perceived in the mouth,—there is a constriction in the throat and gullet, with griping or colicky pains in the stomach and bowels, increased flow of saliva, nausea and vomiting. Blue vitriol is a powerful emetic, and vomiting is rapidly excited by it. The abdomen is distended, the pain in this cavity is increased by pressure, and not relieved by vomiting, and there is purging with tenesmus. The vomited liquids have a bluish or greenish color, and the discharges by the bowels are sometimes greenish, bloody-looking, or dark-colored. These symptoms commence generally in a few minutes after the poison has been taken, and are fully developed within one or two hours. Jaundice and suppression of urine have been observed in some cases. The above-mentioned symptoms are chiefly connected with the *irritant* effects of the salt of copper on the stomach and bowels. When the poison has been absorbed, another set of symptoms, indicative of an action on the brain and nervous system, are witnessed. There is hurried and difficult breathing, with a small quick pulse, great weakness, intense thirst, cold perspiration and coldness of the limbs, headache, giddiness, stupor, coma, tetanic or other convulsions, followed by paralysis of motion or sensation in the limbs. The patient gradually sinks and dies exhausted in a few hours or days. In some cases the symptoms assume at once an entirely nervous character; there are severe headache, great depression, restlessness, trembling of the limbs, cramps, coldness of the surface, small irregular pulse, dilatation of the pupils, with stupor, coma, tetanus, or paralysis. These symptoms are, however, commonly preceded by vomiting, purging, and colicky pains in the abdomen.

Dr. Percival met with a case in which violent convulsions were produced in a girl by a dose of two drachms of sulphate of copper. A woman who had taken five drachms presented after thirty-six hours the following symptoms: Small pulse, contracted features, general uneasiness and suppression of urine. She recovered in ten days after she had taken the poison (Journal de Chimie, 1847, 331). A woman, æt. 32, swallowed seven drachms of the sulphate mixed with three drachms of sulphate of iron. The symptoms of irritation set in with sickness, and continued for twenty-four hours, when she fell into a state of stupor. Some hours after this she was found on her knees retching and vomiting a yellowish liquid, and complaining of pain in the stomach. Any liquid that she took was rejected. In twenty-four hours purging came on: the evacuations were black and yellow in color, and of the consistency of thin gruel: she complained of great thirst and constriction of the throat, severe pain in the stomach and bowels, intense headache, and tremblings of greater or less duration; the vomiting and purging continued until the close of the second day, when the sickness abated. On the morning of the third day, she was so weak that she had not strength to sit up in bed: pain in the bowels and extreme thirst were the most prominent symptoms; and the evacuations were black and watery. (The blackness was probably due to the presence of a salt of iron.) A little brandy was then administered to her,—a greenish-colored liquid was observed to flow from her mouth, and she died about seventy-two hours after taking the poison, without a convulsion, twitching, or sign of pain. (Lancet, Aug. 30th, 1856, p. 248.) In 1836, a girl, sixteen months old, put some pieces of blue-stone, which were given to her to play with, into her mouth. In a quarter of an hour she was observed to vomit a bluish-green-colored matter with pieces of the sulphate of copper in it. The skin was alternately cold and hot, but there was neither purging nor convulsions; the child became insensible before death. She died in four hours from the taking of the poison. These facts are sufficient to show that the sulphate of copper, although it is frequently taken in large doses without producing serious symptoms, should be ranked among poisons. (See ante, p. 21; also, Casper's Vierteljahrschrift, October, 1857, p. 228.)

In forming an opinion from the green color of the vomited matters in alleged cases of poisoning by copper, the practitioner must remember that a morbid state of the bile may give a most vivid copper-green color to liquids thrown from the stomach. I have seen this in a recent case, and from the intensity and persistency of the green color, poisoning was suspected. A slight chemical examination will show whether the color is owing to bile or to a cupreous poison. (See post, Analysis.)

Verdigris produces symptoms somewhat similar to those caused by the sulphate of copper. There is a strong styptic metallic taste, with a sense of constriction in the throat, followed by severe colicky pains,—vomiting of a green-colored liquid, purging, and tenesmus. In a case reported by Pyl, a woman who took *two ounces* of verdigris died in three days: in addition to the symptoms above described, there were convulsions and paralysis before death. Niemann relates that a female, æt. 24, swallowed *half an ounce* of verdigris, and died under symptoms of violent gastric irritation in sixty hours. (Taschenbuch, 458.) In consequence of the great uncertainty of its operation, this compound is not employed as a medicine.

A case of poisoning by this substance, in which the symptoms were accurately observed, is reported in the Edinburgh Medical and Surgical Journal for July, 1844. A woman, æt. 28, swallowed a large dose of verdigris. She was soon afterwards seized with great anxiety, vomiting, acute pains and swelling of the abdomen, sensation of burning heat in the throat, coldness, and severe cramp in the extremities, a laboring pulse, swelling of the face, with the eyes sparkling. An emetic brought away some half-digested food, without any traces of poison. The next morning there was pain in swallowing, swelling of the throat, the abdomen distended and painful on the least pressure, the countenance heavy, the face flushed, and the pulse oppressed. About two pounds of a distinctly-greenish fluid, with some blood, were thrown off the stomach. The symptoms became aggravated; the face and eyelids swollen and red, the eyes prominent, the abdomen drawn in, and the rectum irritable and painful. On the second day there was a tendency to coma, the face was pale, the lips swollen, the gums ulcerated, and there was an abundant discharge of viscid saliva. Purging took place for the first time since the poison was taken; and acetate of copper was detected in the discharges in pretty large quantity. There were several spasmodic fits. On the third day some viscid glairy matter, of a greenish color and tinged with blood, was vomited, and the spasms continued. On the fourth day bleeding from the nose with general cramps came on, and the urine was suppressed. There was coldness of the surface with convulsions. After the lapse of about a week the patient still had vomitings of greenish glairy matters, with uneasiness in the abdomen: but from this date she gradually recovered.

The subchloride or *oxychloride* has thus given rise to accidental poisoning. It is also used as a pigment. The following is a case of poisoning by it. A boy between two and three years of age swallowed part of a small cake of green water-color, such as is sold in the color-boxes for children. Very soon afterwards he was attacked with vomiting and coldness of the extremities. Notwithstanding the exhibition of an antimonial emetic, the symptoms continued to become aggravated, and the child died. (Henke's Zeitschrift der S. A. i, 188, 1844.) This compound of copper is often formed in culinary utensils, and may then give rise to accidents, when food containing common salt has been prepared in the copper vessel without proper precautions. (See Journal de Pharmacie, Juin, 1845, 471.) Prof. Barzellotti relates an instance in which he himself narrowly escaped partaking of the poisonous food. At a monastery near Sienna the monks were one day, soon after dinner, seized with violent symptoms of irritant poisoning. They suffered chiefly from severe pain in the abdomen, nausea, difficulty of passing urine, spasms of the muscles, and trembling of the limbs. Those who were affected with vomiting and purging were speedily relieved; but others, who had no evacuations, suffered from giddiness, headache, intense thirst, and an unpleasant metallic taste in the mouth. Remedies were applied, and they all eventually recovered. It appeared, on inquiry, that the monks were in the habit of keeping their salt-fish in a copper vessel, in which it was dressed for a second day's meal. This vessel was badly tinned; and when the fish was examined, it was observed to be covered with a green jelly, and the sides of the vessel with which the fish was in contact, had a green color. The cause of the symptoms was no longer doubtful: subchloride of copper had been here formed by the action of the salt on the metal. (Quest. di Med. Leg. tomo ii, p. 185.) Several cases of a similar kind are reported by Orfila, i, 619.

A case of poisoning by the *carbonate of copper* occurred to M. Desgranges, of Bordeaux. A man died in about six hours, as it was supposed from the effects of an unknown quantity of this poison which he had taken. When first seen he was insensible; he had sustained some violence from a fall, and there was great coldness of the extremities. There was neither vomiting, purging, nor pain in the abdomen on pressure. (Med. Gaz. xxxi, 495.)

Chronic poisoning by copper.—When the symptoms of acute poisoning have passed away, when the cupreous salt has been taken for a long period in small doses, or the person has been exposed to emanations from copper salts, or alloys, other effects are manifested. The most prominent after-effects are excessive irritability of the alimentary canal, attended with frequent disposition to vomit,—colic, purging, and tenesmus: and there is at the same time loss of appetite, alternations of cold and heat, great prostration of strength, with emaciation, tremors of the limbs, and occasionally paralysis. There is a coppery or metallic taste in the mouth, increased thirst, hot skin, with a small, frequent, irregular pulse. After a few days, there is tenderness with distension of the abdomen, and colicky pains of a severe kind, with symptoms resembling those of dysentery; the evacuations are of a greenish color and mixed with mucus and blood. There is jaundice, with some of the nervous symptoms already described under the head of acute poisoning. The patient sometimes dies from fever and exhaustion. (For a further account of the symptoms and those which accompany copper-colic, see Bocker, Vergiftungen, 1857, p. 42.)

One of the most satisfactory accounts of chronic or slow poisoning by copper, has been published by Dr. Moore. It occurred from want of cleanliness in the use of culinary utensils, and it shows that without great circumspection, a medical man may be completely deceived respecting the origin of the malady affecting many persons simultaneously. On the return of the Indian Coolie emigrants from Guiana to Calcutta, a kind of acute idiopathic dysentery made its appearance in the ship, and it was at first referred to bad water, change of climate, and other causes. Dr. Moore examined the copper-plates on which the fish, rice, and ghee (butter), eaten by the natives, was cooked, and found the surface was coated by a green composition which, when scraped off and examined, proved to be a mixture of chloride and sulphate (?) of copper. The cause of the disease was then apparent. A few hours after taking the meal, the patients complained of violent pains and cramps in the stomach and lower bowels, and there was constant vomiting of greenish and yellowish-green bile. When this was not ejected from the stomach, their sufferings from dry retching were most severe; and the feeling of constriction in the lower part of the chest and along the course of the gullet was still more distressing. Every twenty minutes there was an attempt to evacuate the bowels, but no feculent matter was discharged: blood in small quantities, slimy mucous stools, tinged with blood, shreds of lymph, and frothy ash-colored secretions, were passed from the rectum without affording the patients the slightest relief. Pressure over the abdomen, especially in the pit of the stomach, and in one case, over the arch of the colon, caused severe pain. There were griping pains in the loins and sacrum, at the navel, and in the iliac region, with tenesmus and a burning sensation at the anus. In the commencement of the attack, there was acute fever, pungent heat of the skin, headache, urgent thirst, loss of appetite, prostration of strength, furred and clammy tongue, foul taste in the mouth, with a rapid, small, and wiry pulse. In the more severe cases, there was great depression of the vital powers, the pulse exceedingly rapid and weak, the skin cold, extremities benumbed; the secretion of urine was in a few instances suppressed, in others the urine was retained in the bladder. The symptoms in most instances subsided in eight or ten days under the free use of emetics and castor oil; in others a long time elapsed before the mucous discharges from the alimentary canal and the tenesmus abated,—the disease assuming all the characters of chronic dysentery. One man was subsequently attacked with symptoms of chronic poisoning in an aggravated form, from neglect in the use of a copper-vessel, and sank under the attack. (Lancet, April 11, 1846, p. 414.)

French pathologists have described a copper-colic to which workers in this metal are liable, owing, as it is supposed, to the inhalation of the fine dust of copper or its oxide. According to Orfila, it is in some respects analogous to lead-colic, but it differs from it in being accompanied by a greater degree of irritation in the stomach and bowels. (Toxicologie, i, 912.) The existence of this as an independent form of colic has been denied by some authorities. (Annales d'Hygiène, 1847, i, 392; and Avril, 1858, p. 328. Casper's Vierteljahrschrift, 1852, i, p. 79; 1855, ii, p. 222; 1856, ii, p. 41; and 1857, ii, p. 228.) There is, however, sufficient evidence to establish the existence of this form of copper poisoning. Dr. Corrigan, who has given some attention to this subject, has arrived at the following conclusions: 1. Copper will act as a slow poison, by absorption, undermining the constitution, producing emaciation, catarrh, and loss of strength, and leaving the system in a state little capable of resisting the ordinary exciting causes of many diseases. 2. The symptoms, although not acute, are well marked: they are emaciation, a cachectic appearance, loss of muscular strength, colicky pains, cough, without physical signs to account for it, *and the peculiar characteristic signs of retraction of the gums, with a purple, not a blue edge.* 3. In none of the cases detailed, although there was muscular debility, was there either acute colic with constipation, or the local paralysis that so often results from the poison of lead;—and the color of the gums was quite distinct from that produced by lead. 4. Copper, in chronic poisoning, seems to exert its deleterious influence mainly on the nutritive functions, or assimilation, including absorption and secretion, while lead acts energetically on the nervous system of both organic and animal life, exhibited in its action on the former by producing obstinate constipation, and on the latter by the violent pains of lead colic, as well as by the production of a peculiar form of paralysis. (Dublin Hospital Gazette for Sept. 1854; Lancet, Jan. 1855.)

Effects of external application.—The salts of copper are capable of acting locally, and if applied to a wounded or ulcerated surface, they may become absorbed, and thus affect the system. Sulphate of copper is occasionally used as an escharotic. The solution of this salt, after frequent contact, hardens the unbroken skin, discolors it, and impairs its sensibility. Orfila found that two drachms of acetate of copper, finely powdered, when introduced beneath the cellular membrane of the neck of a large dog, caused death in five days. In another experiment, the same dose applied to the cellular tissue of the thigh, killed the animal in thirty hours (i, 618). Violent phlegmonous inflammation is sometimes occasioned by small quantities of the salts of copper becoming introduced into the system through wounded or abraded surfaces. Mr. Stafford met with a case in which a woman pricked her thumb with a pin. She afterwards scoured out a dirty copper, and her thumb immediately swelled to double its natural size. The whole hand and arm became much swollen and inflamed, and extensive abcesses formed: the patient also suffered from fever, from which she slowly recovered. A second case occurred to the same gentleman, in which severe symptoms followed a puncture produced by corroded copper wire. (Med. Gaz. xxxv, 828.) In these cases the poisonous salt may be the carbonate, subacetate, or subchloride,—most commonly the former. It is probable that the severity of the symptoms may be in some instances ascribed to peculiarity of constitution,—the very small quantity of the salt of copper which can be absorbed scarcely sufficing to account for them.

APPEARANCES AFTER DEATH.—In acute poisoning by the salts of copper, the mucous membrane of the stomach and intestines has been more or less thickened and inflamed in the few fatal cases which have been hitherto examined: the membrane has been also found eroded and softened in poisoning by verdigris. The gullet has presented an inflammatory appearance. In a case of poisoning by verdigris, quoted by Orfila, the stomach was inflamed and thickened, especially towards the pylorus (the intestinal opening), the orifice of which, from the general thickening, was almost obliterated. The small intestines were throughout inflamed, and perforation had taken place, so that part of the green liquid was effused into the abdomen. The large intestines were distended in some parts, and contracted in others, and the rectum was ulcerated on its inner surface. (Toxicologie, i, 623.) In some cases the intestines have been found highly inflamed, perforated, and even in a gangrenous state. The lining membrane of the alimentary canal has throughout presented a deep green color, owing to small particles of verdigris adhering to it. It has been said that this is an uncertain character of poisoning by copper; since a morbid state of the bile often gives a similar color to the mucous membrane of the stomach and duodenum. This objection cannot apply when the green color is found in the gullet, and throughout the intestines: and, under any circumstances, the evidence from the presence of a green color would amount to nothing in the judgment of a prudent witness, unless copper were freely detected in the parts so colored. It is well to remember that the green stain, if due to copper, would be turned blue by ammonia. The liver, stomach, and kidneys have been found congested. In the case of a child poisoned by the subchloride (see ante, p. 462), there was nothing to indicate especially the action of an irritant poison, if we except a slight congestion of the cerebral vessels. The child, it appears, had swallowed about a scruple of the green color. It was remarkable that there was not the least sign of irritation or inflammation in the alimentary canal. Death was ascribed to the exhaustion resulting from violent vomiting; and to a congestion of blood in the brain thereby produced.

In the fatal case of poisoning by the carbonate (ante, p. 462), the mucous membrane of the gullet and stomach was covered with the green-colored compound. The larger end of the stomach was reddened and corroded in patches. The mucous membrane of the intestines as well as the fluid contained in them was of a green color.

The appearances presented in fatal cases of chronic poisoning by copper are well indicated in one of those which occurred to Dr. Moore (ante, p. 463). The mucous membrane of the lower part of the gullet, and that of the stomach between the two orifices, was the seat of extensive and deep-seated inflammation. The shades of red varied from a bright vermilion or scarlet to a deep red or violet color. The patches of a dark red or brownish color were comparatively small and circumscribed, situated in general beneath the mucous membrane of the under surface of the stomach. The membrane in these situations was softened, pulpy but not excoriated, and free from the appearance of having sloughed. At the lesser opening the membrane was intensely inflamed, glistening, and tumid from a quantity of serous fluid deposited beneath the submucous cellular tissue. The mucous membrane of the duodenum and small intestines was also inflamed in irregular patches; and there were traces of inflammation in the large intestines, including the rectum. Eight ounces of a saffron-colored fluid were found in the peritoneal cavity, and on the peritoneal surface of the intestines there were numerous minute spots of inflammatory redness. There was no effusion of lymph or other sign of peritoneal inflammation. (Lancet, April 11, 1846, 414.)

FATAL DOSE.—As the fatal cases of acute poisoning by copper have been but few, it is impossible to assign, with any accuracy, the fatal dose of the salts of this poison. Five drachms of the sulphate have been taken without causing death; and, on the whole, the use of this mineral appears to be more dangerous when taken for some time in small doses, than when a large quantity is swallowed at once. One of the earliest effects on the stomach is the ejection of the substance by vomiting. Böcker assigns the fatal dose at from one to two ounces of verdigris, or blue vitriol; but seven drachms have destroyed the life of an adult. A quantity of subchloride, equivalent to a scruple, or twenty grains, proved fatal to a child (ante, p. 462); and one of the most rapidly fatal cases was that of a child, which died in *four hours* from taking an unknown quantity of blue vitriol. In Reg. v. Smith (Monmouth Lent Assizes, 1856), prisoner was charged with administering blue vitriol to the prosecutor. It was proved that he had put some crystals of blue vitriol into a bottle of cider, and the prosecutor suffered from symptoms of irritation by reason of his having taken a portion. The fatal dose was here made a subject of inquiry, and the medical witness replied, "Half the quantity found in the bottle;" although it is not stated what quantity was found therein. The prisoner was acquitted, on the ground, apparently, that he did not know that blue-stone was a "deadly" poison! The medicinal dose of sulphate of copper, as a tonic, is from one to three or four grains; and as an emetic, from five to fifteen grains. No other preparation of copper is commonly used as an internal medicine in this country.

TREATMENT.—In general there is violent vomiting,—the salts of copper acting powerfully as emetics. The efforts of the stomach should be promoted by the free use of warm water, milk, barley-water, or any mucilaginous drink, and the employment of the stomach-pump. This instrument is of little service when the poison has been taken, as it generally is, in coarse powder. Various antidotes have been proposed. *Sugar* was formerly strongly recommended, on the principle that it had the property of reducing the salts of copper to the state of insoluble and inert red oxide; but this is only under very peculiar circumstances, not likely to be met with in the stomach. (Ann. d'Hyg. 1833, ii, 207.) M. Postel is still inclined to regard it as an antidote, although it seems that animals to which he administered it died; but not *so rapidly* as when the poison was allowed to act by itself! *Albumen* is well known to form an insoluble compound with oxide of copper, provided the albumen be in very large excess; for the albuminate of copper is easily dissolved by an excess of the solution of sulphate; and the albuminate may be itself a poison: still it may reduce the activity of the *soluble* salts of copper, and thus it is advisable to administer albumen both of the yolk and white of egg, conjointly with the other means recommended. There is no objection to the administration of brown sugar, or honey, dissolved in albumen, to aid the chemical change, or a mixture of reduced iron and albumen. On the insoluble salts these antidotes have no action. Dr. Edwards, some years since, recommended the use of *iron filings* for precipitating the copper, and Bouchardat has since recommended "reduced iron;" but the action in this case is slow, and is immediately arrested by the iron becoming enveloped by a thin film of copper. If the iron even precipitated all the copper in the metallic state, sulphate of iron would be formed in the stomach, and this is itself an irritant. Bouchardat has also recommended the use of the hydrated persulphuret of iron, on chemical principles. Castor oil, or emollient injections, may be used.

CHEMICAL ANALYSIS.

The salts of copper are generally known by their color: whether in the solid state or in solution, they are either blue or green. The salts of one other metal are also of a green color,—namely, nickel; but there are striking chemical differences between the salts of this metal and those of copper. There are three *soluble* salts of copper; two of these are blue, the sulphate and nitrate,—and one green, the chloride. The salt should be dissolved in water, diluted, and the following tests may be then applied. The insoluble salts may be dissolved in a diluted acid, and then tested. The solutions of the cupreous salts generally have an acid reaction. The tests for the oxide of copper are: 1. *Solution of ammonia.* This produces a bluish-white precipitate, soluble in an excess of the test, forming a deep violet-blue solution. 2. *Ferrocyanide of potassium*, a rich claret-red precipitate. If the quantity of copper be small, the liquid acquires merely a light red-brown color; if large, the precipitate is of a gelatinous consistency. The ferrocyanide of potassium will act on the violet-blue solution produced by ammonia, provided an acid is previously added (sulphuric) to neutralize the ammonia. One portion of liquid may thus be examined by the two tests. 3. *Sulphuretted hydrogen gas*, or hydrosulphuret of ammonia, gives a deep chocolate-brown precipitate; or, if the copper be in small proportion, merely a brown color, either in neutral or acid solutions. 4. A slip of *polished iron* (a common needle), suspended by a thread in the liquid, is speedily coated with a layer of copper, even when the salt is in small proportion. When much diluted, a drop of diluted sulphuric acid may be added, and the iron allowed to remain some hours or days. The iron will be slowly removed, and a hollow cylinder of metallic copper will remain. This may be dissolved in diluted nitric acid, and tested with the foregoing tests; or the needle coated with copper may be immersed in a solution of ammonia and exposed to air. The liquid then becomes slowly blue, and the nature of the metal is thus clearly established. Half a grain of sulphate of copper dissolved in sixteen ounces of water, may be thus easily detected.

It was proposed by Orfila to substitute *phosphorus* for polished iron. This substance most effectually separates metallic copper from its salts, but it is not so convenient for use as iron.

5. *The galvanic test.*—If a few drops of the copper solution be placed on platina foil, slightly acidulated with a diluted acid, and the platina be then touched through the solution with a thin slip of zinc,—metallic copper, of its well-known red color, is immediately deposited on the platina. Zinc and platina wires twisted round each other may be immersed in the liquid, and allowed to stand some hours, when the proportion of copper is small. Under these circumstances, there is merely a reddish-brown stain on the platina; but a blue liquid is formed by pouring on it ammonia, or it may be dissolved by nitric acid, and tested by 1 and 2. By these tests it is easy to detect the 250th part of a grain, or even less.

There are no *objections* to the inferences from these tests when taken together; but if reliance be placed on one or two only, the analyst may fall into an error. Thus, ammonia produces in a salt of *nickel* a color somewhat similar to that produced in a salt of copper; but ferrocyanide of potassium precipitates a salt of nickel of a pea-green color,—a reaction very different from that produced on a salt of copper. The persalts of *uranium* give with the ferrocyanide of potassium a deep red color, which, in a diluted state, might be mistaken for the effect produced by copper; but ammonia gives a yellow precipitate in a persalt of uranium, and sulphuretted hydrogen and hydrosulphuret of ammonia give a yellow-brown precipitate of uranium. The color of the copper precipitate approaches a dark crimson; that of uranium, a blood-red. When ammonia is added to the uranium precipitate, it is destroyed, and a yellow deposit of oxide of uranium takes place in the liquid: when added to the ferrocyanide of copper, the liquid acquires a bluish-green color. To the action of the third test, when taken by itself, there are many objections; but these are entirely removed by the application of the other tests. The action of iron, and of zinc with platina, is peculiar.

Blue vitriol is soluble in four parts of cold and two of boiling water, and is easily obtained in well-defined rhombic crystals by evaporating a small quantity of the solution on a slip of glass. Nitrate of baryta added to the solution indicates the presence of sulphuric acid.

There are several varieties of *verdigris*, some of which are blue, and others green. It is partially soluble in water; but readily when this is acidulated with acetic or muriatic acid. If a portion of the powder be heated in a reduction-tube, a film of metallic copper is produced, and acetic acid vapor escapes. Acetic acid is, however, readily discovered by boiling the powder in diluted sulphuric acid.

The *insoluble* or partially soluble salts of copper, which may give rise to questions of poisoning, are the subchloride and carbonate. They possess these common characters,—that, 1, when rubbed on a steel spatula with a few drops of diluted sulphuric acid, metallic copper is abundantly precipitated on the *iron;* and, 2, when treated with a strong solution of *ammonia*, they acquire a rich violet-blue color. They are both dissolved by acids; the carbonate with effervescence.

Copper in organic liquids.—The oxide of copper is liable to be precipitated by certain organic principles, as albumen, fibrin, and mucous membrane; but some of these organic compounds are easily dissolved by acids, or even by an excess of the solution of cupreous salt. A portion at least of the salt of copper is, therefore, commonly held dissolved. In such cases, there is one peculiar character possessed by these liquids, —*i. e.* they have a decidedly *green color*, even when the copper salt is in a far less than poisonous proportion.

Separation by iron.—We first filter the liquid through tow, calico, or paper, and save the insoluble portions for a separate operation. We may use, as a trial-test, either a needle, zinc, with platina, or add to it a portion of oxalic acid. The last gives a bluish-white precipitate only when the copper is dissolved in moderately large quantity and the liquid is not very acid. If the needle be not coated with copper in the course of a few hours, it is certain that there is no detectable quantity of the poison present in the liquid. The process by iron will give a good result notwithstanding the presence of a large quantity of organic matter, and in spite of great dilution; by it a very small quantity of a salt of copper may be easily discovered in tea, coffee, porter, or gruel, provided we take care to acidulate the liquid slightly with diluted sulphuric acid before introducing the polished needle. The following is the result of an experiment: One-third of a grain of sulphate of copper dissolved in water, and mixed with four ounces of thick gruel (= 1-6000th part) gave a distinct deposit of copper on a needle in twenty-four hours. Instead of a needle, a coil of the finest bright iron-wire may be used. When the quantity of copper is small, the red color of the deposited metal is not always perceptible: it appears brown or black, and the deposit may be obscured by its being mixed with some oxide of iron. When we are in doubt about the nature of the deposit, the iron wire should be well washed, and then placed in a small quantity of solution of ammonia and exposed to air. The liquid will soon acquire a blue color if any metallic copper is present. By this method a grain of sulphate of copper diffused in sixteen ounces of a decoction of sarsaparilla and water, was easily detected in a few hours, although the cupreous deposit was not rendered perceptible on the iron by its red color.

Separation as a sulphuret.—If the copper salt be present in *large* quantity, any of the trial-tests will indicate it immediately. We destroy the viscidity of the liquid by diluting it if necessary, and pass into it a current of sulphuretted hydrogen gas, in order to precipitate all the copper in a state of sulphuret. The black sulphuret may be collected, washed, dried, and then boiled in equal parts of nitric acid and water for a quarter of an hour. Nitrate and sulphate of copper are produced and dissolved, a fact indicated by the liquid acquiring a rich blue color; and some sulphur is at the same time separated. This liquid, when filtered and diluted, will give the usual reactions with the tests for copper.

Separation by platina.—The following is an expeditious and simple method of obtaining copper from organic liquids. Having filtered the liquid, let a portion of it be placed in a clean platina capsule or crucible. A few drops of diluted sulphuric acid are added, and a slip of zinc foil is then introduced. Wherever the platina is touched by the zinc, metallic copper is deposited; and, after having in this way coated the platina capsule, the surplus liquid may be poured off and the capsule well washed out. A few drops of nitric acid, with a small quantity of water, may be used to dissolve the deposit of metallic copper; and by evaporating the acid liquid and redissolving the residue in water, a pure solution of nitrate of copper is obtained, giving the usual blue color with ammonia; and, when this blue liquid is slightly acidulated with diluted sulphuric acid, it gives a red color with ferrocyanide of potassium. Copper, in moderate quantity, may be thus easily separated from milk, gruel, porter, or the most complex organic liquids.

Detection of copper in the tissues. Absorption and deposition.—Copper, like other metallic poisons, is absorbed and deposited in the tissues. It has been discovered in the blood, organs, and secretions, when its salts have been taken. Orfila has found the metal in the lungs, heart, liver, spleen, and kidneys of animals poisoned by it; but he could discover no traces of it in the blood or urine, although it must undoubtedly be conveyed into the blood. M. Flandin asserts that copper is not found in the heart and kidneys. (Des Poisons, i, 569.) Wibmer, according to Sobernheim, detected copper in the liver of a dog to which he had for several weeks given small doses of the sulphate. Absorbed copper is most easily detected in the liver. About two ounces of this organ have been found sufficient for the experiment. MM. Danger and Flandin have stated that in cases of poisoning, copper may be detected more readily in the bronchial secretion than in the urine. (Ann. d'Hyg. 1843, 452; ante, p. 65.) In a case of poisoning by the carbonate (ante, p. 462), copper was found in the urine, but not in the blood. (Med. Gaz. vol. xxxi, p. 495.)

There may be no poisonous salt of copper dissolved in the organic liquid subjected to analysis,—a fact commonly indicated by the entire want of action on polished iron. The oxide of copper may be intimately combined with some organic principles, or even with the mucous membrane of the stomach itself, and exist only in an *insoluble* form. It will then be necessary to cut up these substances, which commonly have a green or blue color, and boil them for an hour in water containing one-sixth part of strong nitric acid. The acid liquid should be filtered, and evaporated to dryness; and if much organic matter be present, this should be destroyed by redigesting it in strong nitric acid, and again evaporating to dryness. Pure chlorate of potash may be used with the nitric acid, to aid in the entire destruction of organic matter. Water will now dissolve out of the dried residue any copper as nitrate which may have been taken up by the nitric acid. Copper may be also extracted by carbonization with sulphuric acid, or by entirely decomposing the tissue by nitro-muriatic acid. The organic matter may be simply dried, and then calicined in a platina crucible. The ash may be digested in a small quantity of strong hydrochloric acid, and the acid liquid diluted, filtered, and evaporated, to get rid of the excess of acid. A piece of bright iron wire may be kept immersed for a few hours, and occasionally examined: a red film upon the surface will indicate copper. If the liquid be too acid, the iron will be attacked and dissolved, and the object of the experiment frustrated.

It has been objected to the process of calcination, that copper is contained as a natural constituent in most of the organs of the body, and the term *normal copper* has been applied to it. According to Sarzeau, this metal is also present in the incinerated residue of sugar, coffee, madder, wheat flour, and cheese. Blood, milk, and other liquids of the body, in cases where no poisonous salt of copper has been taken, are also said to have yielded it. One chemist made a mixture of eggs, some strong coffee, and bread and butter; he dried and incinerated the mass, and detected copper in the residue! The metal is said to have been found in bread, beef, and mustard. Thus, then, according to this view, copper exists naturally, not only in the organs of the human body, but likewise in some of the most common articles of food. It is, however, very probable that copper may, in some of these cases, have been introduced accidentally during the analysis, and thus have led to an erroneous inference, especially as it was only found in infinitesimal traces. Dr. Christison could not detect any portion of the metal in the animal fluids; and in some experiments on large quantities of oatmeal and bread, I did not detect the smallest portion of copper, although the tests answered perfectly when a cupreous salt was purposely added in minute proportion. MM. Danger and Flandin arrived at the same results, *i. e.* that neither copper nor lead enter into the composition of the healthy human body or of the food of man; and that where they are said to have been detected, their presence must be ascribed to their adventitious introduction in articles of food, or during the analysis. The question is of some interest in toxicology, for it has been already brought forward as an objection to medical evidence. In August, 1843, M. Barse communicated to the Academy of Sciences the results of some analyses made on the bodies of two subjects taken from the hospitals of Paris. They had died from ordinary disease. M. Barse states that he detected copper and lead in both subjects. In September, 1843, M. Rossignon, of Lyons, addressed a note to the Academy of Sciences, on copper as it exists in the organic tissues of many vegetables and animals. M. Rossignon states that he detected copper in all his experiments on the human body: he found it in the blood and muscular fibre of man, in the tissues of many domestic animals (the dog), and in the common vegetables used as food. The gelatin used as soup at the hospital of St. Louis yielded 0·03 per cent. of pure copper. Common sorrel gave two per cent. of neutral oxalate of copper; chocolate from 0·07 to 0·05 per cent. The bread generally used in Paris gave, in 1000 parts of incinerated residue, from 0·05 to 0·08 of copper (fraudulently introduced as sulphate?). Coffee, chicory, madder, and sugar yielded traces of the metal,—in the latter case mixed with lead. Barley-sugar contained copper: and in the sugar of starch it formed four per cent. by weight of the carbonized residue. M. Legrip states, that 1000 parts of liver or spleen contain 0·009 of copper (Böcker, Vergiftungen, 1857, p. 99). These results are directly opposed to those obtained by MM. Danger and Flandin, M. Chevallier, M. Chevreul, and others. M. Chevallier states, that he has sometimes found traces of copper in the non-poisoned body, and at other times not. When Orfila was required to repeat his experiments on the alleged existence of normal copper, before a committee of eminent chemists, it required the incineration of three healthy livers, and digestion of the ash in nitro-muriatic acid, in order to procure any evidence of the presence of copper! (Journ. de Chim. Août, 1847, p. 484.)

Dr. Odling has informed me that he has frequently found traces of copper in the incinerated residue of the organs of the human and animal body. On one occasion he demonstrated to me its presence in a kidney. There had been, so far as it was known, no administration of a salt of copper. It is not improbable, that these conflicting results may be reconciled by the supposition that copper is occasionally introduced into the animal body through certain articles of food which contain it as impurity, and it is thence transferred to and deposited in the organs. The term "normal" cannot therefore be properly applied to copper so received; on the contrary, it is an abnormal constituent of the body which has not undergone the usual process of elimination. As an objection to medical evidence of poisoning, the admission of its occasional presence in the body has no force: 1. Because in poisoning by copper, there would be very few cases in which the whole of the chemical evidence rested on traces of the metal found by an incineration of the viscera: such a case is very unlikely to occur; for chemical evidence is in general abundantly afforded by an analysis of a portion of the poisoned substance swallowed, or of the contents of the stomach. 2. If the only chemical evidence were that derived from incineration, then this could afford no proof of poisoning, unless that fact were already sufficiently made out by symptoms, appearances, and moral circumstances, in which case such infinitesimal proof might be easily dispensed with. In a case of falsely imputed poisoning, it may be said that the detection of copper in a particular article of food, such as bread, would lead a medical jurist into error, since the discovery of this metal in the bread might bear out the imputation, and inculpate an innocent person. This hypothesis does not appear probable. The normal copper, said to exist in food, has not been found to form, according to its discoverers, more than the 100,000th part of the food examined: if the imputation of poisoning were well-founded, and copper were discovered at all, the metal would be in infinitely larger proportion than this, so as to leave no doubt of its actual admixture. These facts furnish an objection, therefore, only to the evidence of those who rely exclusively on the infinitesimal results of a chemical analysis.

It is not probable that a medical jurist would be required to seek for a cupreous poison in a body which had been so long interred that the remains were intermixed with the soil. But it is not the less necessary to state that, according to the researches of M. Walchner, copper, like arsenic, is almost universally found in ferruginous soils, and in most kinds of marls and clays. Wherever the ores of iron exist, there copper will be found: in this way it may be dissolved in water, and percolate through the superficial strata. (See Comptes Rendus, Sept. 21, 1846, 612.) Admitting the truth of this observation, a comparative analysis of the earth of the cemetery would be required in the very rare case in which the decomposed remains of the dead had become intermixed with the soil. M. Walchner simply digested the earth in muriatic acid, and precipitated the copper from the acid solution by a current of sulphuretted hydrogen gas.

QUANTITATIVE ANALYSIS.—This is best determined by converting the salt of copper to the state of black oxide, every 100 parts of which are equal to 312 of crystallized sulphate. If the cupreous salt be precipitated as sulphuret, this may be transformed to black oxide by digestion in nitric acid, and subsequent incineration, or by boiling the salt of copper with an excess of solution of potash.

COPPER IN ARTICLES OF FOOD.—The medico-legal history of poisoning by copper would be incomplete without some remarks on the action of certain articles of food on this metal when used for culinary purposes. This is not an unfrequent form of accidental poisoning. The symptoms are those of chronic poisoning elsewhere described (p. 463).

Water.—Water does not appear to have any marked action on this metal when the surface is clean. On exposing clean metallic copper in contact with *distilled* water and air for a period of forty-three days, the water was clear, without color, taste, or smell, and the copper bright and unchanged as when first immersed. The tests for copper showed that that there was none of the metal dissolved. On evaporating three ounces to dryness, however, a very slight green sediment (less than one-eighth of a grain) was procured, which, on analysis, was found to be carbonate of copper. When *river water* was substituted, similar results were obtained, but the carbonate of copper left on evaporation, was in rather larger quantity. This may have arisen from the presence of bicarbonate of lime. The contact of water with clean copper vessels is therefore not likely to lead to the production of a sufficient quantity of poisonous salt to affect health. When the copper is corroded or dirty, the poisonous crust may become mechanically diffused through the water, and give rise to chronic poisoning. In water containing common salt a greenish sediment is formed (subchloride)—which is quite insoluble, but which if finely diffused through the water may produce all the effects of chronic poisoning.

If the water contain any acid, such as vinegar, the copper is more rapidly oxidized, and the liquid acquires a green color. If the copper vessels be kept perfectly clean, and the food prepared in them be allowed to cool in other vessels, there is not much risk of its acquiring a poisonous impregnation: nevertheless, no acid, saline, fatty, or oily liquid should be prepared as an article of food in a copper vessel. (See Ann. d'Hyg. 1832, i, 102.) Under the influence of heat and air, a portion of copper is dissolved, and the oily or other liquid acquires a green color. The preparation of fruits, such as preserves, in copper vessels, is necessarily attended with some risk; for on cooling, a green crust may be formed on the copper, just above the surface where the air and acid liquid meet. Some substances appear to be but little liable to this impregnation: thus, coffee, beer, milk, or tea has been boiled for two hours together, in a clean copper vessel, without any portion of the metal being taken up by either of the liquids. (See Falconer on the Poison of Copper, 65, London, 1774; also Orfila, i, 611.) Accidental impregnation is usually prevented by lining the copper vessel with tin; but in large boilers this plan cannot always be adopted,—cleanliness alone is trusted to, and this is a sufficient preventive when properly observed. Dr. Paasch attributes many of the cases of alleged poisoning by the use of copper culinary utensils in Germany to the effects of sausage poison (Casper's Vierteljahrschrift, 1852, p. 79).

al effects resulting from the impregnation of *acid liquids* with
re illustrated by the following accident, which occurred a few
ce. A servant-girl at a farmhouse put a copper vessel into a
taining the wash with which the pigs were fed. This is said to
mmon practice in many parts of the country, as the acidity of the
serves to cleanse the copper vessel. A number of pigs were fed
this wash, and six of them died;—their bodies were examined, and
tomachs were found inflamed. Owing to the ignorance which prevails
hese matters, soup and other articles of food, such as acid wine, beer,
cider, are often improperly kept in copper vessels exposed to air, and
us become poisoned. Dupuytren relates that a whole family was poi-
oned by eating crayfish, cooked and allowed to cool in a copper vessel,
o which vinegar had been added. Three persons died. A similar case
occurred to Barzellotti (ante, p. 462). Severe colic and vomiting have
been caused by vinegar placed in copper utensils; and copper stop-cocks
to vessels containing acid liquids may give rise to similar symptoms.
Dr. Moore considers that the attacks of cholera and of acute or chronic
dysentery, under which Europeans arriving in the East Indies so fre-
quently suffer, are in many cases due to the general employment of cop-
per utensils for culinary purposes, and from the want of cleanliness on
the part of the native cooks, who use butter, salt, and acids, without re-
moving the cupreous incrustation which is formed on the surface or in
the rims of the vessel. Hot butter or lard, like hot oil, readily dissolves
copper, forming fatty salts of which oxide of copper is the base.

In the making of preserved *fruits* and vegetable *pickles*, the salts of
copper (blue vitriol) are sometimes used for the purpose of giving a rich
green color. Many of the green pickles sold in shops are thus impreg-
nated with the vegetable salts of this metal, to which they owe their
bright grass-green color. If the fruit or pickle be placed in a solution
of ammonia, and copper is present, the substance is speedily turned blue.
The iron-test is, however, more delicate. A needle immersed in the
pickle, or plunged in the solid, will be speedily coated with copper. The
quantity of copper contained in such articles may not be sufficient to
produce fatal effects; but when frequently taken, serious symptoms of
gastric irritation are sometimes produced, and in infants or children these
may assume an alarming character. (See Falconer, 87.) A few years
since, some preserved gooseberries were sent to me for examination, as
it was suspected, from their having produced symptoms of poisoning in
a child, that they were contaminated with copper. The suspicion turne
out to be correct. The cook had mixed with them some blue vitriol
improve the green color. Dr. Hassall states that he found copper in s
teen different samples of London pickles, and it was most abundan
those which were green. (Food and its Adulterations, 388.)

Bread.—A few years since, a fraudulent practice existed on the
tinent of mixing sulphate of copper with the dough of bread. The
tity of cupreous salt used was small, but still it was a noxious ad
(Ann. d'Hyg. 1830, 342; 1831, 338; 1840, ii, p. 123.) Ac
rimentalists, bread always contains traces of coppe
the blue vitriol with which seed corn is f
imie, 1846, p. 686.) It may also

introduced accidentally during the making of the bread, as where copper utensils have been used for this purpose. Thus it may be found in bread, and not in the flour from which the bread was made, or in the flour and not in the corn. MM. Theulen and Servan having found copper in a specimen of bread, ascertained by further examination that copper cylinders had been used in grinding the corn. A small quantity of oxide falling from these would at once account for the contamination irrespective of fraud. (See Orfila, i, 651; Galtier, i, 607.) Alum, as well as salt, is used in the making of bread, and small quantities of copper sometimes exist in these substances. Copper may be detected in the ashes of bread by the process of incineration.

Medicines.—Copper may be occasionally found as an accidental ingredient in pills or extracts. Brass and bell-metal mortars are used for the preparation or mixture of pills or extracts with mineral substances. The presence of a small quantity of acid in the extract may lead to the impregnation of the medicine with a trace of copper.

Accidental poisoning by copper has occurred from the use of what is called *German silver*, but which should rather be called *white brass*, as it is an alloy of copper and zinc with nickel. Some specimens of this alloy contain fifty per cent. by weight of copper. The following case of poisoning by this alloy occurred in Paris in 1838. A lady, after having had eels for dinner, was awakened in the night by intense headache, followed by nausea, vomiting, and severe colic. These symptoms were removed under proper treatment. Her physician ascertained that the eels had been cooked with butter and vinegar in an earthenware vessel, and he found that the metal spoon, which was of German silver, presented on different parts greenish-colored spots. Chemical analysis showed that a poisonous salt of copper had been thus accidentally produced: a fact proved by polishing the spoon, and then placing it in a hot mixture of bread, butter, and vinegar. Half an hour after the mixture had cooled, green spots were perceived on it; and in twelve hours the spoon was quite green, as well as the butter in contact with it. It is stated that an impure alloy used by some of the lower grade of dentists has been so largely composed of copper, as to have affected the health of those who use the plates for the support of artificial teeth. The acid and salts in the saliva favor the production of a poisonous salt of copper, giving rise to a peculiar metallic taste.

CHAPTER XXXI.

POISONING BY ANTIMONY—TARTAR EMETIC—ITS EFFECTS ON CHILDREN—SYMPTOMS—ACUTE AND CHRONIC POISONING—EXTERNAL APPLICATION—APPEARANCES AFTER DEATH—FATAL DOSE, AND PERIOD OF DEATH—TREATMENT—CHEMICAL ANALYSIS—CHLORIDE OF ANTIMONY—POISONING BY ZINC, TIN, SILVER, IRON, GOLD, AND BICHROMATE OF POTASH.

General remarks.—METALLIC ANTIMONY is not regarded as a poison, but when respired in the state of *vapor*, it is stated to have produced serious symptoms. A case of poisoning by the vapors of antimony is reported in the Edinburgh Medical and Surgical Journal (lv, 265). Orfila suggests that the effects said to have been produced by this metal in vapor may be ascribed to arsenic, which is present in most specimens of crude antimony as it is used in manufactures. (Toxicol. i, 504.) Of the antimonial compounds, there are only two which require special consideration, namely, tartar emetic and chloride of antimony.

TARTARIZED ANTIMONY—TARTAR EMETIC—STIBIATED TARTAR.

Tartar emetic owes its poisonous properties to the oxide of antimony, of which it contains 44 per cent., the residue being composed of potash, tartaric acid, and water. In consequence of its having been frequently given to adults in large doses without causing death, its poisonous properties have been doubted. This subject has already been fully considered (ante, p. 99.) One reason why the symptoms are often so slight from comparatively large doses, is owing to its possessing such violent emetic properties. This leads to the early expulsion of the greater part of the poison from the stomach. When given in small doses at intervals the effects are those of chronic poisoning. Common medicinal doses often produce violent vomiting and great depression. A case is related by Dr. Lambert, in which only four grains of tartar emetic gave rise to violent pain in the abdomen, vomiting, and purging. (See p. 481.)

It appears, from the observations of the late Mr. Goodlad of Manchester, and Mr. Noble, that tartar emetic, even in small doses, is liable to act as a poison on the young. Mr. Wilton records four cases in which prostration and collapse followed the administration of ordinary doses of tartar emetic to young children. Two of them were fatal. Three-quarters of a grain of tartar emetic were prescribed for an infant recovering from measles. The child died in an hour from the depressing effects of the medicine. A similar dose was prescribed for another child of the same parents: violent vomiting and purging supervened, and this case also ended fatally. In a third instance, of a girl, æt. 4, suffering from hooping-cough, one-third of a grain given in divided doses produced alarming symptoms, which rendered a discontinuance of the medicine necessary. (Journal de Chimie, Sept. 1847, p. 471; see also Med. Gaz. vol. xl, p. 351.)

Tartar emetic appears to act more as an irritant than as a corrosive; but the symptoms which it produces, like those of all corrosive poisons, are generally immediate,—some at least are manifested within a few minutes. It is used in medicine both externally and internally. TARTAR EMETIC SOLUTION, or VINUM ANT. POT. TART., is a solution of tartar emetic in sherry wine; it contains two grains in an ounce. It is prescribed in doses of ten drops to one drachm, and in much larger doses as an emetic. It has no taste. Tartar emetic ointment contains one-fifth of its weight of this substance.

SYMPTOMS. ACUTE POISONING—When tartar emetic is taken in a dose of from one to two or three drachms or upwards, the person experiences a strong metallic taste, which continues for some time.

In a few minutes there is nausea followed by violent vomiting, which continues generally until the stomach is cleared, and even for some time afterwards, as a result of local irritation. There is pain in the stomach and bowels, followed by purging, more or less violent; a sense of burning heat and constriction or choking in the throat, extending through the whole length of the gullet to the stomach, difficulty of swallowing, soreness of the mouth and throat, followed by the peeling off of the lining membrane or the formation of aphthous crust, at first whitish, but becoming subsequently discolored, brown and black. When this symptom occurs, it is probable that the same condition of the mucous membrane exists in the gullet, stomach, and part of the intestinal canal. In some cases there is great thirst, with increased flow of saliva. The vomited matters consist of a white stringy mucus, locking up solid portions of the poison, but sometimes tinged with blood or bile — the evacuations are liquid and bilious. There are cramps in the arms and legs; sometimes there are severe tetanic spasms: coldness of the surface, with clammy perspiration, attended with flushing, and a congested state of the head and face, a feeling of extreme depression, loss of muscular power, pulse small, contracted, and feeble,—in advanced cases fluttering, and barely perceptible; respiration short and painful, livid or dusky appearance of the lips and face, especially around the eyes, which are sunk; loss of voice, complete incapacity for any exertion; an eruption resembling that of smallpox occasionally showing itself on the skin; wandering or delirium, with loss of consciousness.

These symptoms are not met with in every case; thus, vomiting and purging may coexist, or one may be vicarious of the other. In certain cases, neither of these symptoms may be present, and then those affecting the nervous system are generally more prominent. The intensity of the symptoms, the rapidity of their progress, and the speedy access of collapse, chiefly distinguish those of the acute from the chronic form. In the latter variety there is nausea, a loathing of food and incessant retching, without actual vomiting until food is taken. The vomited matters are sometimes white (mucus), but at a later period colored with bile, and the symptoms recur with severity after each administration of the antimony in food or medicine,—the prostration of strength being great in proportion to the frequency of this recurrence. There has been noticed a greatly increased secretion of urine. In no instance has suppression been observed, as in cases of arsenical poisoning. Antimony appears to be carried off abundantly by the urine. In the acute form of poisoning, the presence of poison in the food may generally be perceived by the taste; in the chronic form, from the smallness of the quantity, there may be no taste perceptible.

One of the remarkable characters of the acute form is that, in spite of the violence and severity of the symptoms, even when the collapse and depression appear to indicate impending dissolution, there is an astonishing power of recovery. When one large dose only is administered, the case proceeds steadily to recovery or death, generally the former if the case is placed early under proper treatment. In this respect, acute antimonial is distinguished from acute arsenical poisoning. In the latter, in spite of early treatment, and the removal of the whole or the greater part of the poison from the stomach, the case frequently terminates fatally. Should, however, another dose of antimony be taken at or about the time at which recovery is taking place from the effects of the first, it will be easily understood that the person will sink under the effects of the poison. If any doubt exist concerning the cause of the symptoms, *i. e.* whether they be due to bilious cholera or some form of gastritis or gastro-enteritis, then an examination of the urine should be made. If this be examined at intervals, it will be found to contain antimony, should the case be one of antimonial poisoning. An analysis directed to the matters vomited and the excreta, will also aid the practitioner in forming an opinion.

In a case reported by Mr. Freer, a man, æt. 28, swallowed half an ounce (240 grains) of tartar emetic by mistake for Epsom salts, and recovered from its effects. An hour after the poison had been taken, he was found in the following state: his pulse imperceptible; tongue dry and red; countenance cold and livid, bathed with clammy perspiration, and indicative of great suffering; violent pain in the stomach, and over the whole of the abdomen, with constant spasmodic contraction of all the muscles, particularly of the abdomen and arms. The fingers were firmly contracted, and the muscles quite rigid. He vomited only once, about *half an hour* after he had swallowed the poison, and after this he had constant involuntary aqueous purging. An emetic of mustard and salt was given to him, and this produced violent vomiting of bilious matter. Green tea, brandy, and decoction of oak-bark, were freely given. The cramps, vomitings, and aqueous purging, continued for six hours. The symptoms then became mitigated, and he gradually recovered, suffering chiefly from profuse night perspirations. (Lancet, May 22, 1847, 535.) This case is remarkable for the anomalous character of the symptoms, as, in the absence of active vomiting, an emetic was actually required to be given,—also for the recovery of the individual after a very large dose of the poison.

Dr. Gleaves, U. S., has related, in the Western Journal of Medicine and Surgery, the following case: A young man swallowed by mistake a *tablespoonful* of tartar emetic (= about 478 grains). In an hour afterwards he was speechless, pulseless, and apparently dying. Although he drank freely of cold water, and irritated his throat repeatedly with his finger, no vomiting had occurred. During the first three hours he vomited only two or three times, and the matter ejected was chiefly the water taken to favor vomiting. After the lapse of two hours there was violent purging. In seven hours this ceased, and there was great thirst, with a sense of burning pain in the throat, gullet, stomach, and bowels. There was also great irritability of the stomach, and the vomited matters were tinged with blood. On the following day the vomiting continued, but the purging was arrested. The throat was covered with pustules: there was pain in passing the urine, which was copious. On the third day, the whole body was covered with genuine tartar emetic pustules. These began to heal, and the patient to recover, in about two weeks. (Medical Times, Jan. 24, 1846, p. 127.) This is the only case of poisoning by tartarized antimony, in which pustular eruptions on the skin are stated to have been observed. It is otherwise remarkable for recovery from so large a dose, considering that but little of the poison could have been expelled in the first instance by vomiting.

Chronic poisoning.—A good account of the effects produced by this poison, given at intervals in small doses to healthy persons, has been published by Dr. Mayerhofer (Heller's Archiv. 1846, pts. 2, 3, 4, page 100, *et seq.*). The principal symptoms are,—great nausea, vomiting of mucous and bilious liquids, great depression, watery purging, followed often by constipation of the bowels,—small, contracted, and frequent pulse,—loss of voice and muscular strength,—coldness of the skin, with clammy perspiration, and death from complete exhaustion. Several cases have recently occurred in this country, which show that tartar emetic has been thus criminally and fatally used. In addition to the cases of Ann Palmer and J. P. Cook, there are those of Reg. v. McMullen (Liverpool Summer Assizes, 1856), Reg. v. Freeman (Drogheda Spring Assizes, 1857), and Reg. v. Hardman (Lancaster Summer Assizes, 1857). (For an account of these I must refer to Guy's Hospital Reports for October, 1857.)

External application.—Tartar emetic is said to have produced symptoms of irritant poisoning when applied externally to the skin in the form of ointment as a counter-irritant. In a case where the skin was but little affected by the use of this ointment, nausea and sickness were produced, which disappeared when the use of the ointment was discontinued. Although it is extensively used as an external application by medical practitioners, it is rare to hear of cases of poisoning by it under these circumstances. Dr. Griffiths, of Philadelphia, states that, thus applied, it has produced violent salivation. Its effects appear to be limited to the production of local irritation and a pustular eruption on the parts of the skin to which the ointment is applied.

APPEARANCES AFTER DEATH.—The mouth, throat, and gullet have been found inflamed, or in an aphthous state. The inflammation has been sometimes confined to the mucous membrane of the throat and the lower part of the gullet. The mucous membrane of the stomach is more or less reddened in patches or spots, as a result of inflammation,—the membrane is softened or corroded, and easily removed by friction, sometimes covered with false membrane or aphthous crusts; the surface darkened, inflamed, and ulcerated; and small ulcers with pustular exudations are occasionally found. The contents of the stomach are of a dark brownish color, consisting chiefly of mucous matters, colored either by blood, bile, or by a mixture of both. The peritoneal or external coat of the stomach has been found inflamed; the intestines present similar appearances, the inflamed portions of mucous membrane being seen chiefly in the duodenum, cæcum, and rectum; the contents of the intestines are bilious or bloody, with much mucus. There are aphthous ulcerations in the glands of the small intestines; the lungs show more or less congestion in portions of the lobes; the heart is empty, or if blood be contained in its cavities this is dark-colored and liquid; the blood liquid throughout the body. The brain and its membranes have been found congested, and the substance of the brain softened. Cases have been met with in which these appearances have not been found, or the changes were slight and unimportant (Böcker, Vergiftungen, p. 37).

ath hronic poisoning, the liver has been found enlarged, and
its structure was easily broken down. The organs of the
found well preserved. These appearances will neces-
rding to the duration of the case. When life is pro-
nay be the appearances of gastro-enteritis in a severe

n, a boy æt. 5 years, and a girl æt. 3 years, each swal-
r containing *ten grains* of tartarized antimony mixed with
. It was stated that, in twenty minutes after taking the
y were seized with violent vomiting and purging, and great
of strength, followed by convulsions and tetanic spasms:
great thirst. The boy died in eight hours, and the girl
rteen hours, after swallowing the dose. The bodies were
ed r a s ter death. In that of the boy
on of serum r t pleura: the lower lobe of the
riorly was r r than natural, and the peritoneum was
from recent inflammation. The mucous membrane of the duo-
med, and covered with a whitish-yellow viscid secretion;
ed throughout the intestinal canal, although the color was
yellow in the large intestines: there was no ulceration. The
coat of the stomach was inflamed. The mucous membrane of
was much inflamed, especially about the larger curvature, and
c orifice: there was no ulceration. The contents (about
unces and a half of a dark grumous fluid, having a slightly acid
on) were adherent to it; and in one case there was a patch of
pn. The tests used did not indicate the presence of antimony. With regard to other appearances, the tongue was covered with a white fur, and appeared soddened; the fauces were not inflamed; the windpipe and gullet had a natural appearance. On opening the cranium, the dura mater was found congested; the longitudinal sinus contained a coagulum of lymph, and but little blood. The vessels of the surface of the brain were much injected with dark blood, the whole surface having a deep purple color. Every portion of the brain, when cut, presented many bloody points. The cerebellum and medulla oblongata were also congested; there was no effusion in the ventricles, or at the base of the brain. In the body of the girl, the morbid appearances were similar; there were also patches resembling the eruption of scarlatina, on the arms, legs, and neck. The arachnoid membrane was more opaque than usual; and on the mucous membrane of the stomach, where the inflammation was greatest, were two or three white spots, each about the size of a split pea, which appeared to be the commencement of ulceration. (Mr. Hartley, in Lancet, April 25, 1846, 460.)

A girl, æt. 16, swallowed a dose of tartarized antimony, amounting to from forty to sixty grains. There was severe vomiting in a quarter of an hour, and this was soon followed by purging; these symptoms continued for about three hours. She also complained of pain and a burning sensation down the œsophagus. The vomited matters were of a dark color. On the following morning she had recovered from the severity of the symptoms; but in the afternoon there was a relapse. She continually threw her head back and screamed: the skin was warm and moist; the pupils were dilated; and the knees drawn up. She died in about thirty-six hours after taking the poison, and during the six or eight hours previous to her death she was quite delirious. An inspection was made thirty-six hours after death. The throat appeared swollen: the lungs were slightly congested: the heart was healthy, and contained about six drachms of fluid blood. The stomach contained sixteen ounces of a thick bloody fluid: at the greater extremity the coats were softened, and blood was effused under the mucous coat in several places. The small intestines contained a similar fluid with much mucus; but there was no appearance of inflammation. Only slight traces of the poison were found in the contents of the stomach by the usual tests, the greater part having probably passed off by vomiting and purging. (Mr. Beale, in Lancet, Jan. 21, 1854.) In animals poisoned by this substance, it is common to find general inflammation of the alimentary canal.

FATAL DOSE. PERIOD OF DEATH.—The *quantity* of tartar emetic which is actually required to destroy life is unknown. It will probably depend in a great degree on whether active vomiting and purging have been excited or not; for these symptoms have not been present in all cases. Doses of from twenty grains to one ounce have been taken without destroying life; although alarming symptoms of irritation have followed. In one case, related by Orfila, a man, æt. 50, took forty grains of tartarized antimony, and died in about four days. This was the only one out of five cases of poisoning by this substance quoted by Orfila, which proved fatal. (Orfila, i, 480.) Dr. Beck mentions a case in which fifteen grains of this substance in solution killed a child in a few weeks: vomiting and purging ensued, followed by convulsions and death. This case proves that a patient is not always saved by vomiting and purging: the fatal effects, on such an occasion, are probably due to rapid absorption. (See also Medical Gazette, vol. xliv, p. 334.) Dr. Pollock has recorded a case in which an adult was killed in ten hours by a dose of one drachm, in spite of early and violent vomiting. (Med. Gaz. vol. xlv, p. 801.) In two cases observed by Mr. Hartley, which have been elsewhere described (p. 480), *ten grains* killed each child in a few hours. A dose of four grains, however, has been known to produce alarming symptoms. Dr. Lambert, who reports the case in Casper's Wochenschrift, states that this dose gave rise to violent pain in the abdomen, vomiting, and purging. The patient then fell into strong convulsions, which lasted half an hour. He became speechless,—no pulse could be perceived, the skin was cold, and it was supposed that he was dead. Stimulating frictions and poultices were employed, and he slowly recovered in about fourteen days.

The medicinal dose of tartar emetic, in substance, as a sudorific and expectorant, is from 1-12th to 1-8th of a grain: to produce nausea, 1-4th to one-half grain,—to act as an emetic, 1 to 2 grains. In the treatment of inflammatory diseases of the lungs it has been used in much larger doses, although not without dangerous results. (See p. 100.)

Tartar emetic in small doses may occasion death by reason of its exerting a depressing influence on the action of the heart. Aged persons, or those who are debilitated by disease, might die under these circumstances, from a dose or doses which would produce no injury to strong and healthy adults. The effects, however, should be clearly traced to the action of the poison, and not be owing to exhaustion as a result of disease. In February, 1853, Mr. Wakley referred to me for examination a case, in which it was supposed that two doses of antimonial wine, equal to about *three grains* of tartar emetic, had caused the death of a man who was in a diseased condition, by its remote effect upon the heart. No trace of antimony was found in the stomach or tissues, there were no symptoms to indicate poisoning, and, under these circumstances, death could not be reasonably attributed to the medicine. The man died in about twenty hours afterwards, probably from exhaustion of the vital powers as a result of disease, and not from the action of this substance.

Dr. Richardson prescribed for a delicate man, suffering from pneumonia and pleuritis, *one-third* of a grain of tartarized antimony, to be taken every four hours. In half an hour after he had taken the first he suddenly became restless, cold, and faint; he then vomited, and soon afterwards was violently purged. In three hours, although the pain at the chest had disappeared, the vomiting, purging, and prostration were extreme. There was little pulse, the surface was cold; the legs were cramped. The case was similar to one of Asiatic cholera, and was evidently a case of poisoning by antimony. It was found that owing to an accident in dispensing, *three grains* of tartar emetic had been taken by the patient. On another occasion, a stout, active man took fifteen minims of antimonial wine (= 1-16th of a grain of tartarized antimony). This small dose was prescribed, as the patient had informed him that he suffered severely from the effects of antimony. It produced incessant nausea for many hours. There was abdominal pain, with griping, faintness, general exhaustion, and great prostration of strength. There was no purging. (Lancet, April 12, 1856, p. 400.) At p. 101, I have recorded a case in which the 1-24th part of a grain produced alarming symptoms. It has been hitherto supposed that the cases in which this poison has proved fatal have been but few; but I have elsewhere reported thirty-seven, of which sixteen were fatal. The smallest fatal dose was in a child,—*three-quarters of a grain*, and in an adult, two grains; but in this case there were circumstances which favored the fatal operation of the poison (Guy's Hospital Reports, Oct. 1857).

Taking the facts hitherto collected, it appears probable, that under circumstances favorable to its noxious operation on the system (indicated by failure of pulsation and collapse), a dose of from *ten* to *twenty grains*, taken at once, might destroy an adult, and if taken in divided doses, a smaller quantity than this might suffice. Large doses are very uncertain in their operation. In two instances persons have recovered after taking quantities twice, and even eight times, as great as that which has proved fatal to a healthy man. In the Association Medical Journal for April 1, 1853, at page 281, will be found reported a case in which a physician took half an ounce of tartar emetic by mistake for Rochelle salts. Vomiting did not come on for half an hour; but under good medical treatment he recovered in a few days. In another case, fifty-five grains caused the death of an adult in sixteen hours. In one instance a small dose of this substance proved fatal by producing intestinal hemorrhage. (See the same Journal, June 10, 1853, p. 513.)

In reference to the *fatal dose*, it is rather to the *effects* produced than to the *actual quantity* swallowed, that we are to look. As vomiting and purging generally occur speedily, and the poison is known to be ejected, it would be irrational to assume that the dose swallowed remained unaltered in the body. It is important, therefore, not to fix the fatal dose by actual weight. A quantity which may destroy an infant, will not destroy an adult female, and a dose which may kill a delicate woman, or an old person, might not act fatally on a strong and healthy man. A person laboring under disease may be more easily destroyed than one who is healthy, and lastly, there is that ever-varying condition of idiosyncrasy, in which, as it is well known, there is a state of constitution that renders a person more liable to be affected by antimonial compounds, than other individuals apparently in precisely the same conditions as to health, age, &c. Then, again, a dose of ten grains administered at once, may not be attended with the same amount of danger to life as the same quantity given in small doses over many days or weeks.

TREATMENT.—This consists in promoting vomiting by the free administration of tepid water, mixed with butter, oil, or grease,—as well as of milk, or other diluents. The stomach-pump may also be used. Any vegetable infusion containing tannic acid, such as strong green tea, decoction of oak bark, or Peruvian bark, may be given. This principle combines with oxide of antimony to form a compound insoluble in water; and if attended with no other benefit, it at least suspends the operation of the poison. This tannate of antimony is said to be inert; but it is easily dissolved by some vegetable acids (tartaric). Should the decoction not be at hand, Peruvian bark may be given, either in the form of tincture or powder. Cases are reported in which this treatment has been attended with the most decided benefit.

CHEMICAL ANALYSIS.

Tartarized antimony as a solid.—In a state of powder.—1. Tartar emetic is easily dissolved by water,—it is taken up by fourteen parts of cold, and two of boiling water; the solution has a faint acid reaction, and an acrid caustic taste; it is decomposed by long keeping. It is insoluble in alcohol. 2. The powder treated with sulphuretted hydrogen water or hydrosulphuret of ammonia, acquires an orange or reddish-brown color, and is thereby known from other poisonous metallic salts. 3. When heated in a reduction-tube, it is charred, but does not melt before charring, like the acetate of lead. The metal is partially reduced by the carbon of the vegetable acid, and the decomposed mass has a grayish-blue metallic lustre. I have not found that a metallic sublimate is produced in this experiment by the heat of a spirit lamp. Heated on mica in air it is charred and evolves an odor of burnt tartaric acid. 4. When boiled with hydrochloric acid and metallic copper, a gray deposit of antimony takes place on that metal. The color of the deposit is violet, if the quantity be very small, and the deposit is black and pulverulent if very large.

Tartarized antimony in solution.—1. On slowly evaporating a small quantity on a slip of glass, it will crystallize in tetrahedra or in derivatives of the octahedron. If obtained from a very diluted solution, this crystallization is confused, and resembles that of arsenic. 2. *Diluted nitric acid* added to the solution, if moderately concentrated, throws down a white precipitate (subnitrate of antimony): the other two mineral acids act in a similar manner; but, as they precipitate numerous other metallic solutions, there are objections to them which do not hold with respect to nitric acid. The white precipitate thus formed possesses the remarkable property of being easily and entirely redissolved by a solution of tartaric acid: it is also soluble in potash, and in a large excess of nitric acid, so that if much of this acid be added at once, no precipitate is produced. The potash solution, when treated with nitrate of silver, gives a copious black precipitate insoluble in ammonia. 3. *Ferrocyanide of potassium* does not precipitate the solution, whereby tartarized antimony is known from most other metallic poisons. 4. *Hydrosulphuret of ammonia* or *sulphuretted hydrogen gas*, produces in the solution a reddish orange-colored precipitate, differing in color from every other metallic sulphuret. The dry precipitate is soluble in hydrochloric acid, and the solution gives a white precipitate when added to water (see infra). This is the special test for antimony. It will reveal the presence of 1-500th of a grain of tartar emetic, or, under favorable circumstances, the 1000th part of a grain of antimony. There are no objections to the results obtained by this test.

The foregoing tests, it will be observed, merely indicate the presence of oxide of antimony,—but this is in reality the poison which we have to seek,—the cream of tartar with which it is combined being merely the vehicle; and in a case of poisoning, this is no more the object of medico-legal research than if it were the vehicle for the administration of arsenic or corrosive sublimate. It is, besides, well known that the tartar emetic is the only salt of the oxide of antimony in a soluble form which is likely to be met with in medicine or chemistry. Should it be required to prove the presence of cream of tartar, this may be done by filtering the liquid from which the oxide of antimony has been entirely precipitated by sulphuretted hydrogen gas. On evaporating this liquid, the cream of tartar may be obtained.

In liquids containing organic matter.—Tartar emetic is precipitated by tannic acid in all its forms, but not readily by albumen or mucous membrane; therefore it may be found partly dissolved in the liquids of the stomach, provided no antidote has been administered. The organic liquid should be filtered, and then strongly acidulated with tartaric acid. A current of sulphuretted hydrogen gas is now passed into it, until there is no further precipitation. The sulphuret is collected, washed, and dried. If it be the sulphuret of antimony, it will have an orange-red or brown color, and will, when dried, be dissolved by a small quantity of boiling muriatic acid (forming sesquichloride of antimony) with the evolution of sulphuretted hydrogen. The boiling should be continued for several minutes. On adding this solution to a large quantity of water, a dense white precipitate of oxychloride of antimony (powder of Algaroth or Algarotti, *Mercurius Vitæ*) falls down. This is characteristic of antimony. If it be objected that nitrate of *bismuth* undergoes a similar change when poured into water, hydrosulphuret of ammonia will easily enable the operator to distinguish the two metals; the antimonial precipitate is turned of an orange-red color by that solution, while the bismuthic precipitate is turned of a deep black. The antimonial precipitate is easily dissolved by tartaric acid, that of bismuth is not.

A medical jurist must remember that the discovery of tartarized antimony in the contents of a stomach is by no means a proof of its having been taken or administered as a poison; since it is frequently prescribed as a medicine, and often taken as such by persons of their own accord. We could only suspect that it existed as a poison, or had caused death, when the quantity present was large, and there were corresponding appearances of irritation in the alimentary canal. The presence of any quantity, if not lawfully administered as a medicine, is always a suspicious fact, and demands explanation. In two cases of criminal administration in small doses, the quantity found in each body did not exceed three grains. The discovery of it in a medicinal mixture would not of itself be evidence of an intent to poison.

If sulphuretted hydrogen gas fail to show the presence of antimony in an organic liquid, then we must resort to the process described below for the detection of antimony in the tissues.

Quantitative analysis.—The quantity of tartar emetic present in a liquid, may be determined by the weight of the washed and dried sulphuret of antimony: one hundred parts of the dried sulphuret (sesquisulphuret) by weight, are equal to 202·78 parts of crystallized tartar emetic.

Antimony in the tissues. Absorption and deposition.—The facts connected with the absorption, deposition, and elimination of antimony have been already fully described (ante, p. 56). I have found, in accordance with the statements of Orfila, that in poisoning by tartar emetic, antimony is retained by the organs partly in a form soluble in water. A few ounces of the liver or other organ are cut into small pieces, and placed in a mixture of one part of pure hydrochloric acid (first proved to be free from antimony) and seven parts of water. The mixture is boiled, and while boiling, a small piece of thin copper foil, freshly brightened, or a piece of fine copper gauze, may be introduced. Sooner or later, according to the quantity present, antimony is deposited on the copper, producing a gray deposit, with a reddish-violet, or purple tint, if the quantity be small, or iron gray or black, if comparatively large. If no deposit is observed at first, the whole of the liquid must be boiled down on the copper before the inference is drawn that antimony is absent. If the copper be removed without any metallic tarnish or deposit upon its surface, there is no antimony present. If it has acquired a metallic deposit, then, after well washing and drying it, further steps must be resorted to in order to determine that it is really antimony with which the copper is coated. Reinsch considered that antimony was sufficiently indicated: 1, by the color of the deposit being violet; and 2, by the copper foil (when heated) yielding no distinct crystalline sublimate like arsenic; but it was long felt by chemists that these characters, affirmative and negative, were not sufficient for medical evidence. In some researches on the subject in 1855, I found that by deflagrating the coated gauze by small portions, with a small quantity of fused nitrate of soda or potash in a platina or porcelain capsule, that the deposit was immediately removed from the copper; antimoniate of soda was formed if the deposit was antimonial; and by acidulating the liquid with hydrochloric acid and passing into it a current of sulphuretted hydrogen, hydrated sulphide of antimony was procured. A better method of corroboration has been suggested by Dr. Odling. (Guy's Hospital Reports, October, 1856.) The copper with the supposed antimonial deposit is boiled in a small quantity of water, rendered feebly alkaline by pure potash, and colored of a light crimson or pink tint, by the addition of a few drops of a weak infusion of permanganate of potash. In a short time, the copper loses the whole of the metallic deposit; the liquid becomes colorless, and a brownish substance (hydrated peroxide of manganese) falls down, which should be separated by filtration. A few drops of hydrochloric acid are added to the filtered liquid, and a current of sulphuretted hydrogen passed through it. If the deposit were antimonial, antimoniate of potash would be first formed, and the antimony thrown down in the last stage as hydrated orange-red sulphide. Mr. Watson, of Bolton, has since suggested that the coated copper should be boiled in a weak solution of potash only, the metal being partly exposed to air by drawing it out of the alkaline liquid, and then again returning it. In this manner the antimony is gradually oxidized by the air in contact with an alkaline solution, and antimoniate of potash is formed. In about five or ten minutes the copper will have lost the deposit, and the liquid may then be filtered, acidulated with hydrochloric acid, and treated with sulphuretted hydrogen. The orange-red sulphide of antimony, of its characteristic color, is thrown down either immediately, or on allowing the liquid to stand for a few hours. This corroborative test for Reinsch's process can be universally adopted, as potash is procurable when the permanganate may not be forthcoming. The precipitated sulphide obtained, may be too small to allow of the subsequent solution in hydrochloric acid, and precipitation by water; but this additional corroboration is not necessary under the circumstances. Potash may contain oxide of lead, and hydrochloric acid may contain antimony. These liquids should therefore be tested, and their purity clearly ascertained before they are employed for the purpose above mentioned. The permanganate of potash should also be tested in order to exclude the possibility of antimony being introduced by the chemical reagents,—a proof rigorously insisted on when the quantity found is very small.

The presence of antimony as an impurity in hydrochloric acid is not common. In the year 1856, a sample of this acid was sent to me, which contained so much antimony, that on mixing it with water, it gave an abundant precipitate of oxychloride. The acid had been employed with carbonate of soda in making unfermented bread, and it had produced in a provincial town a large amount of sickness, the cause of which could not at the time be explained.

Although antimony is not found to be a *normal constituent* of the healthy human body, it is obvious that by this and other channels, it may occasionally find its way into the system. Hence the great importance of not relying too much on the chemical evidence of its presence in small quantity, when there is no knowledge of symptoms preceding death, or of appearances in the dead body.

Marsh's process has also been employed in the search for antimony in the tissues. A part of the liver or other organ is cut into small pieces, and mixed with about half its weight of concentrated and pure sulphuric acid. The mixture is then heated to carbonization. The dry carbonized residue is treated with a small quantity of pure and strong hydrochloric acid. This acid liquid, drained from the carbonized mass, is introduced into Marsh's apparatus, and the gas proceeding from it tested. 1, Previous to kindling, the antimonuretted hydrogen, if allowed to pass on a solution of nitrate of silver on paper, blackens it, and sets free metallic silver; 2, when kindled, it burns with a pale, lemon-colored flame, evolving a white smoke (oxide); 3, a piece of cold glass acquires, when placed so as to intercept the flame about its centre, a deep coal-black deposit, with gray rings; the bright metallic lustre of antimony appearing on the reverse side of the glass. The coal-black color distinguishes this deposit from that of arsenic, which is hair-brown; but a better corroborative test is to pour on the deposit a few drops of sulphide of ammonium, and evaporate slowly to dryness. If the deposit be owing to antimony, an orange-red stain is left on the glass, not easily dissolved by a weak solution of ammonia. (See ante, p. 355.)

This is said to be a more delicate process than that of Reinsch. Assuming that this is the case, it may be observed, that in medico-legal researches, it is not so much delicacy as *certainty* which is required for evidence; and certainty is abundantly supplied by Reinsch's process, up to very minute fractions of a grain. Beyond this, whether Marsh's or any other process be relied upon, it is not desirable to go.

Detection after long periods.—Antimony, even in a soluble form, does not readily disappear from a dead body after interment. If a person dies with absorbed or free antimony in his body, some portion of the metal may be extracted, probably so long as the viscera remain. In the case of Ann Palmer, the body having been exhumed after an interment of *fourteen months*, antimony was found in the free state in all parts of the alimentary canal, and in the absorbed state, more or less in all the organs. One ovary alone yielded the fiftieth part of a grain. The antimony had partially undergone a chemical change, as a result of putrefaction. In the stomach, a portion had been converted to orange-yellow sulphuret, which dyed the coats in a streak or stain from the inside to the outside. In the rectum it was also partially changed to sulphuret. In this case, antimony manifested an antiseptic property like arsenic, for all the parts in which the metal was found were well preserved. In the case of Ann Bacon, whose body was exhumed in 1857, after *twenty-one months'* interment, antimony was found in the intestines. The presence of the metal was here traced to some small doses of antimonial medicine which had been given to the deceased during her last illness, and shortly before her death.

The plan adopted by MM. Millon and Laveran for the detection of antimony in organic liquids (the urine) and solids, consists in the immersion of a freshly scraped bar of *pure* tin into the cold liquid, acidulated with about one-tenth part, by measure, of pure hydrochloric acid. Antimony, if present, is deposited on the tin in a black powder. (Comptes Rendus, 1856, xxi, p. 637; Annuaire de Chimie, 1846, p. 715.) There is no deposit with arsenic under similar circumstances. This may suffice for pathological but not for medico-legal purposes. In the cases of antimonial poisoning hitherto examined, the quantity of the poison found in a *free* state in the contents of the stomach and bowels, has been only a minute fractional proportion of the quantity swallowed. The amount of *absorbed* antimony found deposited in the organs is always small. In general, from one grain to three or four grains would probably be the whole amount that would be separated by chemical processes from those organs and parts of the body which are usually submitted to analysis for absorbed antimony. (See ante, p. 184.)

Arsenic in antimonial compounds.—The crystals of tartar emetic occasionally contain traces of arsenic. If any antimonial preparation has been given medicinally to a person alleged to have died from arsenic, and traces only of arsenic are discovered in the dead body, a medical witness must be prepared for this objection to chemical evidence. Two persons were tried in France for a double murder by poisoning with arsenic; and arsenic and antimony were detected in the exhumed bodies. The presence of antimony was accounted for by the fact that each of the deceased had taken before death an antimonial quack-medicine. An objection was made to the evidence, that the arsenic might have been mixed with the antimony as an ordinary adulteration in the medicine, none of which could be procured. This view, however, was immediately set aside by the fact that the arsenic was in very large, and the antimony in very small proportion. It is obvious that such an objection could only hold *cæteris paribus* when the arsenic was in minute quantity. (Gaz. Méd. Janvier, 1846.) This question might easily arise in England under the medicinal use of James's powder, antimonial wine, or even of tartarized antimony, given as an emetic to remove a poison. Two cases have recently occurred in this country in which, although antimony was believed to be the cause of death, minute traces of arsenic were found associated with antimony in one of the organs of the body. The source of this contamination is to be traced to the presence of arsenic in the proportion of from 1-60th to 1-20th of its weight in all the German and French sulphide of antimony. Metallic antimony contains from 1-50th to 1-200th part. The crystals of tartarized antimony which are first formed are free from this impurity; but according to Martius, the larger crystals which are principally formed in the mother-liquid contain arsenic. (Gmelin's Handbook of Chemistry, vol. iv, p. 317.) The use of arsenical sulphuric or hydrochloric acid in the preparation of the compound may also prove an additional source of contamination. Orfila attributes some of the severe symptoms occasionally produced by antimony to this contamination with arsenic. (Traité de Toxicologie, i, 616, 5me ed. 1852.)

CHLORIDE OF ANTIMONY—SESQUICHLORIDE OR BUTTER OF ANTIMONY.

This is a highly corrosive liquid, varying from a light yellow to a dark red color: in the latter state containing generally a large quantity of iron. It is a powerful poison, but it is not often taken as such. Orfila mentions only one, and that a doubtful instance, which occurred nearly two hundred years ago. I have collected reports of four cases, in three of which recovery took place, while the other was fatal.

Symptoms.—In 1836, a boy, æt. 12, swallowed by mistake for ginger-beer four or five drachms of a solution of butter of antimony. In half an hour he was seized with vomiting, which continued at intervals for two hours. There was faintness, with general weakness and great prostration of strength. Remedial means were adopted, and the next day the chief symptoms were heat and uneasiness in the mouth and throat, with pain in swallowing. There were numerous abrasions on the mucous membrane of the mouth and fauces; and there was slight fever, from which the boy quite recovered in about eight days.

In a second case, which occurred in 1841, about a tablespoonful of chloride of antimony was given, by mistake for antimonial wine, to a boy æt. 10. Immediately on drinking it, the boy seemed choked: his features were set, and he was unable to speak for some minutes. He vomited freely,—gruel was given to him, which was rejected: he complained of great pain in his throat. Medical assistance was sent for, and about two hours after swallowing the poison, the child labored under the following symptoms. The features were pale and collapsed, the eyes sunk,—the pupils dilated and inactive,—the skin cold,—the mouth filled with a thick tenacious transparent mucus,—nausea, vomiting,—pulse 80 and small, and breathing heavy. He was in a kind of stupor, from which he could, however, be roused to answer questions rationally. He felt a severe burning pain in the throat, extending to the stomach,—increased by deglutition. Under active medical treatment, these alarming symptoms were removed; on the following day it was observed that there were patches of a bright scarlet color in the throat, with difficulty of swallowing. In the course of a few days the boy recovered. The third case occurred to Mr. Bancks, of Stourbridge. On the 16th November, 1846, a boy, æt. 7, swallowed two drachms of chloride of antimony, sent in mistake by a druggist, who immediately discovered his error and applied for medical assistance. There was excoriation of the mouth and throat; the skin was cold and clammy; pulse small and accelerated; burning pain in the stomach, with swelling of the abdomen, and incessant vomiting. Magnesia diffused in water, a decoction of bark and strong tea, were given at intervals until 8 P.M., when there appeared much less pain in the stomach. The boy gradually recovered, and in four days was out of danger. For the next few days he continued to improve, and was soon in perfect health again. It is worthy of remark that the child had taken no food on the morning he swallowed the poison,—a circumstance much against the chance of recovery. (Prov. Med. Journ. Dec. 23, 1846.) Another case of recovery from a dose of an ounce is reported in the Lancet, Feb. 26, 1848, p. 230.

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The only fatal case which I have met with, was communicated to me by Mr. Mann, of Bartholomew Close. An army surgeon swallowed, for the purpose of suicide, from two to three ounces by measure of chloride of antimony. About an hour afterwards, he was seen by Mr. Mann. There was entire prostration of strength, with coldness of the skin, and incessant attempts to vomit. Severe griping pains were felt in the abdomen; and there was a frequent desire to evacuate the bowels, but nothing was passed. In the course of a few hours reaction took place, the pain subsided, and the pulse rose to 120. There was now a strong disposition to sleep, so that he appeared as if laboring under the effects of a narcotic poison. In this state he continued until he died,—ten hours and a half after he had swallowed the poison.

Appearances after death.—The interior of the alimentary canal, from the mouth downwards to the middle of the small intestines, presented, in the above-mentioned case, a black appearance, as if the parts had been charred. In general, there was no mucous membrane remaining, either on the stomach or elsewhere; only a flocculent substance, which could be easily scraped off with the back of the scalpel, leaving the submucous tissues and the peritoneal coat. All these parts were so soft that they were easily torn with the fingers.

Treatment.—The free exhibition of magnesia in milk, as well as of substances containing tannic acid.

CHEMICAL ANALYSIS.

If any portion of the chloride be left in the vessel, it may be tested by adding a few drops to a large quantity of water, when the whitish-yellow oxychloride of antimony will be precipitated; the supernatant liquid will contain muriatic acid, which may be detected by nitrate of silver. It has been already observed, that the only objection to this mode of testing, is, that the salts of *bismuth* are also decomposed by water; but the precipitate in this case is insoluble in tartaric acid, and is blackened by hydrosulphuret of ammonia; while in the case of antimony, it is soluble in that acid, and is changed to an orange-red color by the hydrosulphuret. The precipitated oxide of antimony is soluble in potash; and on adding nitrate of silver to the potash-solution, a dense black precipitate is formed insoluble in ammonia. Nitrate of silver added to the chloride gives a mixed precipitate of chloride of silver and oxide of antimony,—the former being soluble in ammonia. If the chloride contain much iron, the color of the precipitate will be obscured. Ferrocyanide of potassium has no effect on a solution of tartar emetic, but it precipitates the chloride of antimony of a yellow-white; or if much iron be present, Prussian blue is abundantly thrown down.

Organic liquids or solids.—The chloride, as a corrosive, combines with the animal tissues. The antimony may be separated in such cases by boiling them in muriatic or nitro-muriatic acid. By this process, the organic matter will be decomposed and entirely destroyed,—the antimony being recovered on evaporation to dryness, and treating the residue with strong hydrochloric acid. The liquid may then be further tested by the process of Marsh or Reinsch.

A case of attempted poisoning by the administration of the GLASS

(vitrefied oxysulphuret) OF ANTIMONY in soup, is reported by Chaussier. There appears to have been no doubt that some of the substance was swallowed: the man was seized after the meal with pain in the bowels and vomiting. He died in nineteen days. On inspection, no appearances indicative of poison were met with in the alimentary canal, and an opinion was given that the deceased had died from malignant fever. (Mémoires et Rapports, 330.)

PREPARATIONS OF ZINC—SULPHATE OF ZINC—WHITE VITRIOL OR WHITE COPPERAS.

Symptoms and appearances.—The symptoms produced by an overdose of sulphate of zinc are pain in the abdomen and violent vomiting, coming on almost immediately, and followed by purging. After death, the stomach has been found inflamed. The sulphate appears to act as a pure irritant; it has no corrosive properties. This salt may cause death indirectly as the result of exhaustion from violent vomiting, when an ordinary dose has been given to a person already debilitated by disease. (Med. Times and Gaz. July 16, 1853, p. 78.) Dr. Gibb has reported a case of poisoning by this substance, in which a lady recovered after taking sixty-seven grains. (Lancet, May 17, 1856.)

Tommasini relates, that a lady swallowed, by mistake, one ounce of sulphate of zinc. Among the first symptoms were violent pains in the stomach, vomiting and convulsions, in fact, severe irritation. The pain continued for some time. There was paleness of the countenance, coldness of the limbs, irregular pulse, cold sweats, and fainting. This state of collapse was alarming. Stimulants were given. At night, besides pain in the abdomen and unnatural heat, there was fever with other symptoms of reaction. Inflammation of the stomach supervened. It was some time before recovery took place. (Della nuova dottrina med. Italiana, 1817, p. 57.)

Treatment.—Tepid water, with milk or albumen, should be freely given to promote vomiting. Any infusions containing tannic acid may be employed, such as tea, oak-bark, or Peruvian bark. If the poison should have entered into the intestinal canal, a fact indicated by severe pain in the lower part of the abdomen, emollient enemata may be administered.

Chemical analysis.—The pure sulphate is seen in white prismatic crystals, closely resembling in appearance sulphate of magnesia and oxalic acid; from oxalic acid it is distinguished by remaining fixed when heated on platina foil,—from the sulphate of magnesia, by tests applied to its solution. It is readily dissolved by water, this fluid taking up about one-third of its weight at common temperatures. Analysis of the *solution.*—The solution in water has a slightly acid reaction. The following tests may be used for the detection of oxide of zinc. 1. *Ammonia* gives a white precipitate, soluble in an excess of the alkali. 2. *Sesquicarbonate of ammonia*, a white precipitate, also soluble in a large excess of the test. 3. *Ferrocyanide of potassium*, a white precipitate. 4. *Sulphuretted hydrogen* and hydrosulphuret of ammonia, a milky-white precipitate, provided the solution be *pure* and neutral, or nearly so. If the solution be very acid, sulphuretted hydrogen produces no effect whatever. Nitrate of baryta will serve to indicate the presence of sulphuric acid.

In organic mixtures.—If the sulphate of zinc be dissolved, a current of sulphuretted hydrogen gas may be passed into the solution; the presence of zinc is immediately indicated by a milky-white froth—the sulphuret may be collected, and decomposed by boiling it with muriatic acid. The solution may be then tested for zinc.

In the tissues.—If the salt of zinc be decomposed, and we have to search for it in the mucous membrane of the stomach, or in the substance of the liver, it may be cut up and boiled in strong nitric acid. The viscera may be also incinerated with flux, and the zinc procured in the metallic state, or dissolved out of the residue by muriatic acid.

CHLORIDE OF ZINC.

The chloride of zinc is sold to the public as a disinfectant, under the name of "Sir W. Burnett's Fluid." This is a highly concentrated solution of the pure or sometimes impure chloride of the metal; it has been taken by accident in several cases, and in one instance was supposed to have been criminally administered as a poison. It is either colorless like water, and in this state has been fatally mistaken for "fluid magnesia," or yellowish colored from the presence of oxide of iron.

In one case it was taken by mistake for pale ale, and caused alarming symptoms. On two occasions it has caused death by its having been administered by mistake for "fluid magnesia." In August, 1856, it gave rise to a fatal accident in one of the American steamers. It was served to the Rev. T. Marsh, a passenger, by mistake for mineral water. It is stated that he did not swallow more than a mouthful, as he immediately perceived a burning sensation in his throat. He died from the effects on the fourth day. Most of these accidents have occurred from carelessness in keeping this noxious fluid in ordinary wine or medicine bottles, and in proximity to innocuous liquids which resemble it.

Symptoms.—The symptoms are immediate. In a case reported by Dr. Stratton, about two ounces of a solution containing only twelve grains of the chloride were swallowed. The patient immediately felt pain and nausea; vomiting followed, and she recovered, but suffered from some indisposition for three weeks. In a second case, a wineglassful, equivalent to at least two hundred grains of solid chloride, was swallowed. The man instantly experienced burning pain in the gullet, burning and griping pain in the stomach, great nausea, and coldness. Vomiting came on in two minutes; the legs were drawn up to the body; there was cold perspiration, with other signs of collapse. The man perfectly recovered in sixteen days. (Ed. Med. and Surg. Journal, Oct. 1848, p. 335; and British American Journal, Dec. 1848, p. 201.) Other cases show that the concentrated liquid has a strong corrosive action locally, destroying the membrane of the mouth, throat, gullet, and stomach. There has been frothing of the mouth, with general lividity and coldness of the skin. In a case in which only a mouthful, *i. e.* from four to six drachms of the fluid, had been swallowed, the patient experienced giddiness and loss of sight, with immediate burning heat in the stomach: vomiting and purging came on, and the former symptom continued for a week. There was so much irritability of the stomach for a period of three weeks, that the patient became greatly reduced. Among the early symptoms was loss of voice, which did not return for five weeks. (Med. Times, Oct. 11, 1851, p. 382; and Nov. 8, 1851, p. 497.) Dr. R. Hassall has reported a case in which the nervous symptoms were strongly marked, and were of a peculiar kind. Three ounces of "Burnett's Fluid" were swallowed. There was immediately a sense of constriction in the throat, with a hot burning sensation in the stomach. It is worthy of remark, that there was no pain in the mouth, and there was no appearance of corrosion in the cavity or on the lips. There was incessant vomiting, the vomited matters consisting of thick mucus streaked with blood, and some portion of mucous membrane was discharged. There was no purging until the third day, when the discharges from the bowels had a coffee-ground appearance. After the lapse of a fortnight, a train of nervous symptoms set in, indicated by a complete perversion of taste and smell. The patient appeared to have recovered in about three months. (Lancet, Aug. 20, 1853, p. 159.)

Mr. Allanson, of Sheffield, communicated to me the following case, which occurred in July, 1854. A woman, æt. 28, swallowed an ounce of a strong solution of the chloride, which had been sold to her as a disinfecting fluid. In two hours she was lying on her back in a state of great excitement. The face was flushed, the eyes were turned, a frothy saliva was issuing from the mouth, the hands and feet were cold, and the pulse was scarcely perceptible. She was perfectly conscious, and complained of a burning sensation in the mouth, throat, and stomach. The tongue was found swollen, and the mucous membrane red, but there was no excoriation. It was at first supposed that she had swallowed oil of vitriol. In spite of treatment, she died in *four hours* after she had taken the poison. While she survived there was frequent vomiting, but the most prominent symptom was severe pain in the stomach and throat.

Appearances after death.—Out of ten cases of which I have collected the particulars, there have been six deaths. In one, an infant, æt. fifteen months, the lining membrane of the mouth and throat was white and opaque. The stomach was hard and leathery, containing a liquid like curds and whey. Its inner surface was corrugated, opaque, and tinged of a dark leaden hue. The lungs and kidneys were congested. The fluid of the stomach was found to contain chloride of zinc. (Med. Times, July 13, 1850, p. 47.) A seaman, æt. 21, swallowed about half a pint of Burnett's solution. The case proved fatal in spite of treatment, and the appearances (twenty-five hours after death) were as follows: The neck was swollen, the hands were clenched. There was great lividity of the body and arms. The stomach externally was reddened; the mucous membrane was of a deep purple color, and partially corroded and destroyed. The pyloric or intestinal opening was constricted, and the mucous membrane at this part looked as if caustic had been applied to it. The upper part of the gullet was constricted and of a purplish color; the mucous membrane of the remainder covered with a fine false membrane, with a loose coagulum of recently exuded lymph in the centre.

The upper part of the small intestines (duodenum) was of a purplish color, and intense red for about six inches. There were some red patches in the jejunum, and small emphysematous patches near the lower part of the small intestines. The lungs were congested, and the right lung contained a bloody serum. The heart was normal; the ventricles were filled with dark coagula of blood. In the head, the membranes (dura mater) contained more blood than usual, and there was a general congestion of vessels on the surface of the brain, as well as at the base. The gray matter was darker than usual, and the right choroid plexus was enlarged. (Dr. J. Rose, Lancet, Sept. 12, 1857, p. 271.)

In Mr. Allanson's case (supra) the body was examined fifty-three hours after death. The stomach was much distended, of a pale leaden hue, and the veins were dark and prominent. The under surface of the liver, where it was in contact with the stomach, had the same appearance. The intestines and the other viscera were healthy. The stomach contained a quantity of fluid. The coats were of the consistency of thin tripe, and were much thickened at the intestinal end. None of the fluid taken had passed through the pylorus. Well-marked papillæ of a dirty white color covered the whole mucous membrane. The gullet was much inflamed. The epithelial membrane was white and easily detached, appearing like a false membrane. The lungs and heart were perfectly healthy. Chloride of zinc was detected in the stomach.

These facts show that the concentrated solution of chloride of zinc is both a corrosive and an irritant poison, exerting also a powerful action on the nervous system. In a case which proved fatal at Guy's Hospital in 1856, the coats of the stomach were excessively thickened, and had a leathery consistency. The chloride of zinc may destroy life either by producing stricture of the gullet, or by its chemical action on the lining membrane of the stomach, leading to a loss of power of digestion, emaciation, and exhaustion.

Treatment.—The free use of albumen and emetics.

Analysis.—The chlorine may be detected by nitrate of silver; the zinc by the tests above described. (See p. 491.) All the samples which I have examined, with one exception, contained iron. The properties of this liquid became a subject of inquiry in Richards v. Cocking (Guildhall Summer Sittings, 1858). The plaintiff charged the defendant, a druggist, with having supplied him with Burnett's fluid in place of fluid magnesia. There was medical evidence that the plaintiff had suffered from such symptoms as chloride of zinc would produce, and he obtained a verdict. On this occasion I examined the liquid usually sold as Burnett's fluid. I found that it had a sp. gr. of 1·494; that it had an oily consistency, and was frothy when shaken. It coagulated albumen even when much diluted, and strongly corroded animal matter. One fluid-ounce of it by measure contained 372 grains of solid chloride of zinc. It is a most dangerous substance, and is liable to be fatally mistaken for numerous innocent medicines.

In the tissues.—Chloride of zinc may be detected in the tissues by the process of incineration in a porcelain capsule, and digestion of the ash in water, or by carbonizing the organ by nitric acid. It is important to bear in mind that this salt is occasionally used for embalming or preserving dead bodies; hence the discovery of it is of itself no proof of poisoning.

CARBONATE OF ZINC (CALAMINE).

This compound does not appear to have any poisonous action; and it would probably require to be given in large quantity to produce any effect. Carbonate of zinc is the white substance which is formed on the metal when long exposed to air and moisture. Its effects may become a subject of investigation as a matter of medical police, since zinc is now much used for roofing, and also in the manufacture of water-pipes and cisterns. (See Ann. d'Hyg. 1837, 281, ii, 352; also Edinburgh Monthly Journal, Aug. 1850, p. 181.)

PREPARATIONS OF TIN.

The only preparations of this metal which require to be noticed as poisons are the *Chlorides* or *Muriates*, a mixture of which is extensively used in the arts, under the name of *Dyer's Spirit*. The salts may exist in the form of whitish-yellow crystals; but more commonly they are met with in a strongly acid solution in water. They are irritant poisons; but so seldom used as such, that only one death occurred from them in England and Wales during a period of two years.

PREPARATIONS OF SILVER.

Nitrate of Silver. Lunar Caustic. Lapis Infernalis.—This substance, which is commonly met with in small sticks of a white or dark gray color, is readily soluble in distilled water; in common water it forms a milky solution. It acts as a powerful corrosive, destroying all the organic tissues with which it comes in contact. There are at least two cases on record in which it has proved fatal in the human subject: one of these occurred in 1837–8, but the particulars are unknown. The symptoms come on immediately, and the whitish-flaky matter vomited is rendered dark by exposure to light. The presence of dark-colored spots on the skin will also indicate the nature of the poison.

PREPARATIONS OF GOLD.

Perchloride.—This is the only preparation of gold which requires notice. It is a powerful irritant poison, acting locally like the nitrate of silver. Nothing is known of its effects on the human subject; but in administering it to animals, Orfila has found extensive inflammation, and even ulceration, of the mucous membrane of the stomach. (Toxicologie, ii, 80.) The metal is absorbed and carried into the tissues, but its poisonous action appears to be wholly independent of absorption.

PREPARATIONS OF IRON.

Sulphate of Iron. Copperas. Green Vitriol.—This compound has been several times administered with malicious intention. One death from it took place in 1837–8. It cannot, however, be a very active preparation; for a girl who swallowed an ounce of it recovered, although she suffered for some hours from violent pain, vomiting, and purging. (Christison on Poisons, 506.) Green vitriol or copperas is sometimes given as an abortive. A suspicious case is reported, in which a woman far advanced in pregnancy, but enjoying good health, was suddenly seized at midnight with vomiting and purging, and died in about fourteen hours. The body, which had been buried, was disinterred, and iron was found in large quantity in the viscera. The symptoms are not always of this violent kind. In a case which occurred to M. Chevallier, a man gave a large dose of sulphate of iron to his wife. There was neither colic nor vomiting. The woman lost her appetite, but ultimately recovered. In another case reported by the same authority, a woman was tried and convicted of poisoning her husband with sulphate of iron: but in consequence of the great diversity of opinion among the scientific witnesses at the trial respecting the poisonous properties of this mineral salt, and the dose in which it would be likely to operate injuriously, the court and jury recommended that the sentence of death should not be carried into execution. (Ann. d'Hyg. 1851, i, p. 155.) The reader will find some additional remarks in reference to the action of the sulphate of iron on the body, by the late M. Orfila, in the same Journal, 1851, ii, 337.

External application.—A case which seems to show that this substance may really act through the skin, has been reported by Mr. Moore, of York. A healthy boy, æt. 14, after having been employed in picking crystals from the vat in which sulphate of iron was set to crystallize, was attacked with headache and sickness. He vomited several times, felt pains in the calves of his legs, and colicky pains in the abdomen: at the same time his limbs became contracted. The boy had previously complained that the liquor of the crystals, into which he was constantly dipping his hands, had cracked his fingers. In the course of a week or ten days, these symptoms disappeared under treatment. (Med. Gaz. xxx, 351.) No other cause could be assigned for this singular attack, than the frequent contact of the hands with a saturated solution of the green sulphate of iron.

Chemical analysis.—This substance is generally met with in crystals of a sea-green color. It is readily soluble in water. 1. *Ferrocyanide of potassium* added to the solution, gives a greenish-blue precipitate, becoming of a deep blue by exposure to the air. 2. *Hydrosulphuret of ammonia* gives a black precipitate. 3. Nitrate of baryta will show the presence of sulphuric acid.

Muriate of Iron. Tincture of Sesquichloride of Iron.—This is an acid solution of perchloride of iron with alcohol. It is of a deep red color, and is much used in medicine. Dr. Christison relates an instance in which a man by mistake swallowed an ounce and a half of this liquid: the symptoms were somewhat like those produced by muriatic acid. He at first rallied, but died in about five weeks. The stomach was found partially inflamed, and thickened towards the lesser end. This salt has been much used for criminal purposes in France. (See Medical Gazette, vol. xlvii, p. 307: also Ann. d'Hyg. 1850, i, p. 180, 416; and, 1851, i,

p. 155, ii, 837.). A case was reported to the Westminster Medical Society, in November, 1842, in which a girl, æt. 15, five months advanced in pregnancy, swallowed an ounce of the tincture of muriate of iron in four doses in one day, for the purpose of inducing abortion. Great irritation of the whole urinary system followed; but this was speedily removed, and she recovered. Another case of recovery from a large dose of this preparation has been reported by Mr. Amyot. A healthy married female swallowed, by mistake for an aperient draught, *one ounce and a half* of the tincture of muriate of iron. She immediately ejected a portion, and violent retching continued for some time. There was great swelling of the glottis, with cough, and difficulty of swallowing. These symptoms were followed by heat and dryness of the throat, with a pricking sensation along the course of the gullet and stomach; and in the afternoon a quantity of dark grumous blood was vomited. The motions were black, owing doubtless to the action of sulphur upon the metal. In about a month the patient was perfectly restored to health. (Provincial Journal, April 7 and 21, 1847, 180.) Another case of recovery from a large dose has been reported by Sir James Murray. The patient, æt. 72, swallowed by mistake *three ounces* of the tincture in a concentrated state. The tongue soon became swollen; a ropy mucus flowed from the mouth and nose; there was croupy-respiration, with a sense of impending suffocation. The pulse was feeble, the skin cold and clammy, and the face swollen and livid. A castor-oil mixture brought away inky evacuations, and the patient rapidly recovered. (Dub. Med. Press, Feb. 21, 1849.) This liquid has been used in large doses for the purposes of criminal abortion.

Chemical analysis.—The chlorine may be detected by nitrate of silver and nitric acid, while the peroxide of iron is immediately indicated by a precipitate of Prussian blue on adding a solution of *Ferrocyanide of potassium.*

PREPARATIONS OF BISMUTH.

Subnitrate of Bismuth. Pearl-white. Magistery of Bismuth.—This substance, in a dose of *two drachms*, caused the death of an adult in nine days. There was burning pain in the throat, with vomiting and purging, coldness of the surface, and spasms of the arms and legs,—also a strong metallic taste in the mouth. On inspection, the throat, larynx, and gullet were found inflamed; and there was inflammatory redness in the stomach and throughout the intestinal canal. (Sobernheim, 335.) In a case mentioned by Dr. Traill, a man took by mistake *six drachms* of the subnitrate, in divided doses, in three days. He suffered from vomiting and pain in the abdomen and throat, but finally recovered. (Outlines, 115.) These cases are sufficient to prove that a substance very slightly soluble in water may exert a powerfully poisonous action on the human system.

PREPARATIONS OF CHROMIUM.

Bichromate of Potash. Symptoms.—Well-observed instances of poisoning by this compound, which is now extensively used in the arts, are rare; and therefore, the details of the following case, communicated to the Medical Gazette (xxxiii, 734) by Mr. Wilson of Leeds, are of practical interest. A man, aged sixty-four, was found dead in his bed twelve hours after he had gone to rest; he had been heard to snore loudly during the night, but this had occasioned no alarm to his relatives. When discovered, he was lying on his left side, his lower limbs being a little drawn up to his body; his countenance was pale, placid, and composed; eyes and mouth closed; pupils dilated; no discharge from any of the outlets of the body; no marks of vomiting or purging, nor any stain upon his hands or person, or upon the bed linen or furniture. The surface was moderately warm. Some dye-stuff, in the form of a black powder, was found in his pocket. *Appearances.*—On inspection, the brain and its membranes were healthy and natural; there was neither congestion nor effusion in any part. The thoracic viscera were equally healthy, as well as those of the abdomem, with the exception of the liver, which contained several hydatids. A pint of a turbid inky-looking fluid was found in the stomach. The mucous membrane was red and very vascular, particularly at the union of the greater end with the gullet; this was ascribed to the known intemperate habits of the deceased. In the absence of any obvious cause for death, poison was suspected; and on analyzing the contents of the stomach they were found to contain bichromate of potash. The dye-powder taken from the man's pocket consisted of this salt mixed with cream of tartar and sand. It is worthy of remark that there was neither vomiting nor purging. The salt does not appear to have operated so much by its irritant properties as by its indirect effects on the nervous system. This, however, is by no means an unusual occurrence, even with irritants far more powerful than the bichromate of potash. A case has been communicated to me by Mr. Bishop of Kirkstall, in which a boy recovered from the effects of a dose of this salt only after the lapse of four months. The first symptoms were pain, vomiting, dilated and fixed pupils, cramps in the legs, and insensibility. His recovery was due to early treatment. (See Guy's Hosp. Reports, Oct. 1850, p. 214.)

There can be no doubt that bichromate of potash is an active poison. Mr. West has published a case from which it appears that a medical man, who had inadvertently tasted a solution of it, suffered from severe symptoms resembling those of Asiatic cholera. (Provincial Journal, Dec. 24, 1851, p. 700.) Dr. Baer of Baltimore has reported the following case. A man, in drawing off a solution of the bichromate by a syphon, accidentally received a small quantity into his mouth. In a few minutes he perceived great heat in the throat and stomach, and this was followed by violent vomiting of blood and mucus. The vomiting continued incessantly until his death, which took place in *five hours.* On dissection, the mucous membrane of the stomach, duodenum, and about one-fifth of the jejunum, was destroyed in patches. (Beck's Med. Jur. 823.) In this instance, the salt acted as an irritant.

Treatment.—Besides emetics, carbonate of magnesia or chalk, mixed up in a cream with water, should be given.

Chemical Analysis.—This is an acid salt, easily known from all the other metallic poisons, by its crystals having a deep orange-red color.

It is readily soluble in water, and the solution has the rich orange-color of the salt. It has an acid reaction. It may be identified by the following tests: 1. *Nitrate of silver* precipitates the solution of a deep red color. 2. *Acetate of lead* precipitates it of a bright yellow. 3. *Sulphuretted hydrogen gas* produces with it a dingy green precipitate. Potash may be discovered in it by the action of chloride of platina.

SALTS OF PLATINA, PALLADIUM, AND OTHER METALS.

The salts of PLATINA, PALLADIUM, IRIDIUM, RHODIUM, OSMIUM, COBALT, NICKEL, MANGANESE, CERIUM, and URANIUM, also possess an irritant action, partly depending on the acids with which they are combined. According to the experiments of Gmelin on animals, the OXIDE OF OSMIUM appears to be the most active poison among them. They are products of art not met with in common life; and, so far as I can ascertain, they have never been taken as poisons by man. It is unnecessary, therefore, to occupy space by detailing the chemical processes whereby they may be identified; these will be found fully described in all works on chemistry. This concludes the history of the MINERAL IRRITANT POISONS.

VEGETABLE AND ANIMAL IRRITANTS.

CHAPTER XXXII.

MODE OF ACTION OF VEGETABLE IRRITANTS—SAVIN—SYMPTOMS AND APPEARANCES—ITS ACTION AS AN ABORTIVE—CROTON SEEDS AND OIL—COLCHICUM—HELLEBORE—CANTHARIDES, OR SPANISH FLIES—SYMPTOMS—APPEARANCES—FATAL DOSE—ANALYSIS.

THE poisonous substances of an irritant nature which belong to the vegetable and animal kingdoms, are very numerous as a class; but it will here be necessary to notice only those which have either caused death, or given rise to accidental poisoning. The true *vegetable* irritants, soon after they are swallowed, produce severe pain in the abdomen, accompanied by vomiting and purging. There are rarely any cerebral symptoms, and no convulsions.

It must be admitted, however, that the operation of many of them is by no means clearly defined. Stupor, delirium, and convulsions are occasionally observed as secondary effects: hence the distinction between some vegetable irritants and those which are assigned to the neurotic class is purely arbitrary. Further experience may hereafter lead to a better knowledge of their *modus operandi*, and to an improved classification. One circumstance is worthy of remark. The effects of neurotic poisons can commonly be traced to the presence of a poisonous alkaloid in the vegetable. Among the irritants, the effects appear to be principally due to the presence of an acrid oil or resin. There are two, the

Delphinium Staphysagria and *Colchicum Autumnale*, in which alkaloidal principles have been found.

Some of the vegetable irritants act especially on the bowels, and, in mild doses, are safely used as purgatives. In large doses they produce violent purging, and in old and young persons are apt to cause death by exhaustion. There are, however, but few instances recorded of their fatal action on the human body; and the little that is known concerning their operation as poisons, is chiefly derived from the experiments performed by Orfila on animals. The changes found after death are confined to irritation and inflammation of the alimentary canal. These substances (if we except SAVIN) are rarely resorted to by the suicide or murderer,—for large doses are required, and their fatal operation even in these cases is rendered uncertain by the circumstance that they excite vomiting, and are then commonly expelled from the stomach.

Treatment.—In cases of poisoning by the vegetable irritants, emetics and purgatives (castor oil) or injections should be freely employed, and when the poisonous vegetable is expelled, antiphlogistic measures may be used.

SAVIN—JUNIPERUS SABINA.

This is a well-known plant, the leaves or tops of which contain an irritant poison in the form of an acrid volatile oil of a peculiar terebinthinate odor. They exert an irritant action, both in the state of infusion and powder. They yield by distillation about three per cent. by weight of a light yellow oil, on which the irritant properties of the plant depend. The powder is sometimes used in medicine in a dose of from five to twenty grains. Savin is not often taken as a poison for the specific purpose of destroying life; but this is occasionally an indirect result of its use, as a popular means for procuring abortion, and it therefore demands the attention of a medical jurist. From cases which have been referred to me, I believe that poisoning by it is much more frequent than is commonly supposed.

Symptoms and appearances.—From the little that is known of its effects, savin acts by producing violent pain in the abdomen, vomiting and strangury. Purging is not so common an effect as with other irritants. Salivation is sometimes present. After death, the gullet, stomach, and intestines, as well as the kidneys, have been found either much inflamed or highly congested. There is no proof of its having any action as an abortive, except, like other irritants, by causing a violent shock to the system, under which the uterus may expel its contents. Such a result can never be obtained without placing in jeopardy the life of the woman; and when abortion follows, she generally falls a victim. On the other hand, a female may be killed by the poison without abortion ensuing. Out of four fatal cases of the administration of savin and other drugs for the purpose of procuring abortion, the mother died undelivered in three, and in the fourth instance, the child died after it was born. When the vomiting and purging are very severe, abortion may be expected to follow.

The strong local irritant properties of the leaves, which depend on the essential oil, are well known, from the uses of savin-ointment in pharmacy. The plant grows extensively in country places, and is easily accessible to the evil-disposed. It does not appear to have attracted much notice on the continent, for Orfila is silent on poisoning by this substance, except in so far as it affects dogs. Two cases of its fatal effects in the human female were communicated to Dr. Christison. In one, a dose of the strong infusion was twice taken by a female for inducing abortion. She suffered from severe pain and strangury, aborted, and died five days afterwards. On inspection, there was extensive peritoneal inflammation, with the effusion of fibrinous flakes; the inside of the stomach was red, with patches of florid extravasation. The contents had a green color, and savin was proved to be present by the aid of the microscope. In the second case a girl was seized with violent colicky pains, vomiting, straining, difficulty in passing urine, and fever. After suffering several days, she died. The stomach and intestines were inflamed; the former was in some parts black, and at the lower curvature perforated. A greenish powder was also found in this case, and when washed and dried, it had the pungent taste of savin.

Although it is not considered that savin has a direct tendency to produce abortion, it appears, from its therapeutic employment in chlorosis and amenorrhœa, to affect the uterus. The dried powder, which, owing to the loss of volatile oil, is less energetic than the fresh tops, is given in doses of from five to fifteen grains. The medicinal dose of the essential oil is commonly from two to six drops. The infusion and decoction, which are sometimes used for the expulsion of worms, are less energetic than the fresh tops, because they cannot be prepared without giving rise to a loss of the volatile oil. The oil is not so irritant as it is commonly supposed to be; but in those cases in which it has been said to produce no injurious effects in large doses, it is very probable that it was much adulterated.

A well-marked case of poisoning by the tops of savin was referred to me for examination, by Mr. Lord, in May, 1845. The deceased, a healthy female, had reached about the seventh month of pregnancy. She was very well on the Friday, but was seized with vomiting on the Saturday: she stated that she had taken nothing to produce it. The vomiting continued throughout Sunday, and was of a green color. She was first seen by a medical man on Sunday evening. The symptoms were those of inflammation of the stomach and bowels, with great anxiety, and the pulse 150. The green color of the vomited matter was at first supposed to be owing to bile. The vomiting appears to have continued at intervals, but it does not seem that there was any violent purging. Labor came on on Wednesday. The child was born living, but soon died: the female died on the Thursday, *i. e.*, five days after having taken the poison, for there was no proof that any savin could have been taken after Saturday. On inspection, the brain was healthy, the lungs were healthy, except that the air-tubes had a dark red color, the heart flabby: the blood was generally fluid. The lining membrane of the gullet was reddened, and had on it ecchymosed patches. One half of the mucous membrane, from the cardiac orifice upwards, presented a dark red arborescent injection, with slight patches of ecchymosis: there was no erosion or ulceration. In the stomach a large patch of redness, about three inches in length, extended from the greater curvature towards the pylorus. The vessels of the mucous membrane were considerably injected, forming infiltrated patches, especially about the lesser curvature, extending towards the cardiac end; but there was no ulceration or erosion. The stomach contained nearly eight ounces of a greenish fluid, of the appearance and consistency of green-pea soup. By examining a portion of the washed vegetable substance under a microscope, and by drying a portion, rubbing it, and observing the odor, clear evidence was obtained that the green color was owing to the diffusion of finely triturated savin-powder. The interior of the duodenum, especially towards the pylorus, was intensely inflamed, being of the color of cinnabar. Patches of inflammation were found throughout the other portions of the intestinal canal. There was some inflammation of the peritoneum, chiefly of the upper part of the intestines and omentum. The kidneys were inflamed, and of a dark red color—the bladder was healthy. A green-colored mucous matter containing savin, was found in the duodenum, but not in the lower part of the intestines. (Med. Gaz. xxxvi, p. 646.) The quantity of poison taken by the deceased could not be ascertained, but it must have been large. I estimated the quantity remaining in the stomach after five days, under frequent vomiting, at from twenty-five to thirty grains.

In a case which occurred to Mr. Newth, the patient, a pregnant female, eight hours after she had taken savin, was found lying on her back perfectly insensible, and breathing stertorously. She had been suddenly seized with vomiting, and this continued for some time. At first the case was thought to be one of puerperal convulsions. Labor came on, and she died in about four hours, during a fit of pain. She appeared to be between the seventh and eighth month of pregnancy, and the child was born dead. On inspection, twenty-four hours after death, the brain was found gorged with black fluid blood. The stomach was paler than usual, excepting in one or two spots, which were red, as if blood had been effused into the mucous tissue. It contained four ounces of an acid liquid of a brownish-green color. This, on distillation, yielded an opaque liquid, from which a few drops of a yellow oil were separated by means of ether. Some sediment found in a bottle presented, under the microscope, the characters of powdered savin. (Lancet, June 14, 1845, 677.) There can be no doubt that this was the cause of death. The action of the poison appears to have been, in the first instance, like that of an irritant, and just before death like that of a narcotic.

Analysis.—When the poison has been taken in the form of decoction or infusion, it is beyond the reach of chemical tests. The fact of poisoning can then only be elucidated by the symptoms and by circumstantial evidence. If the oil has been taken, it may be separated by distillation, and obtained by agitating the distilled product with one-third of its bulk of ether. Perhaps the most common case is that where the *powder* has been taken. In the cases of Dr. Christison and of Mr. Lord, it will be observed that in spite of great vomiting, some of the powder remained in the stomach for a period of five days. The contents have generally the appearance of green-pea soup. That the color is not owing to bile may be proved by diluting a portion with water, when the green chlorophyll, from its insolubility, will subside in a dense insoluble stratum, whereas, if the color were due to altered bile, the whole of the liquid would remain colored. By washing the green matter in water, and drying it on plates of glass or mica, evidence may be obtained under a good microscope, by the rectilinear course of the fibres and the turpentine cells, that the substance belongs to the coniferæ. The only other poison of the coniferous order is the yew (*Taxus baccata*), but the leaf of this tree differs from that of savin in having a lancet-shaped apex, while savin has a sharply acuminated point. A portion of the green powder dried and well rubbed will give the peculiar odor of savin. When freed from organic matter, it yields, by distillation with water, the essential oil of savin.

OIL OF SAVIN.—This oil is of a light yellow color, and it has a powerful terebinthinate odor, sufficiently peculiar to render this an easy means of identification. A greasy stain made by this oil on paper is entirely dissipated by heat, or only a slight trace of resin is left. It is lighter than water, but insoluble in it, giving to it, however, its odor and an acid reaction. It forms a milky solution with rectified spirit, but a clear transparent solution with ether. It is very soluble in ether, and by this menstruum it may be separated from watery liquids, as the ether floats with it to the top. Nitric acid in the cold, slowly gives to the oil a dark red-brown color.

CROTON-SEEDS AND OIL.

Symptoms and appearances.—The Croton-seeds owe their poisonous properties to the presence of an acrid oil. One or two grains of the *seeds*, when swallowed, are sufficient to produce severe pain in the abdomen, with copious watery motions. Even the dust of the seeds, when inhaled, has caused alarming symptoms. Dr. Pereira mentions the following case: A man had been occupied eight hours in emptying packages of the seeds, and had thus been exposed to the dust. He first experienced a burning sensation in the nose and mouth, tightness in the chest, effusion of tears, and pain in the pit of the stomach. He then became giddy, and fell down insensible. When admitted into the London Hospital, the man was in a state of collapse, complained of burning in the stomach, throat, and head, and of swelling and numbness of the tongue. The region of the stomach felt hot and tense, the pupils were dilated, the breathing short and hurried, pulse 85, and the skin was cold. He complained of pain in the epigastrium for several days; but it is singular that there was no purging. (Mat. Med. ii, pt. 1, p. 406.)

The *oil* has a hot burning taste. One or two drops are commonly sufficient to produce pain in the abdomen, and purging; but Dr. Traill states that a female patient usually took three drops for a dose without inconvenience,—an effect of habit. (Outlines, 149.) In one case a teaspoonful was given by mistake, to a child æt. 4, who had previously eaten a full meal of bread and milk. In five minutes, the child was seized with violent vomiting and purging, and these symptoms were soon followed by alarming prostration. Under the use of warm fomentations, and of milk and mucilage, the child recovered in two days. (Dr. Cowan in Prov. Trans. N. S. 1, p. 121.) The recovery was here probably due to the oil having been taken on a full stomach and to early vomiting. Dr. Cowan states that he has known similar symptoms to have been produced in an adult by *half a drop* of the oil. In large doses, the pain is described as hot and burning, extending from the mouth downwards; there is violent vomiting with purging, and the patient sinks exhausted. After death, the alimentary canal is found inflamed. Even the endermic application of the oil is stated in some cases to have produced severe symptoms, although, according to Dr. Buchanan, it acts only as a local irritant. (Medical Gazette, xxxix, 671.) A case occurred in Paris, in 1839, in which a man swallowed by mistake two drachms and a half of croton oil. In three-quarters of an hour, the surface was cold and clammy, the pulse imperceptible, respiration difficult, and the extremities and face were as blue as in the collapsed stage of cholera. In an hour and a half purging set in; the stools were passed involuntarily, and the abdomen was very sensitive to the touch. The patient complained of a burning pain in the course of the œsophagus. He died in four hours after swallowing the poison; and it is singular that there was no marked change in the mucous membrane of the stomach. (Orfila, Tox. i, 108.) In June, 1855, a patient in the Dumfries Infirmary swallowed by mistake about three drachms and a half of a liniment containing croton oil. He became aware of his mistake immediately after he had committed it, and, in a few minutes thereafter, he experienced a violent burning sensation, extending from the throat to the stomach, and he had also violent pain in the stomach. He complained of a spasmodic suffocative feeling, and convulsively gasped for breath for several minutes. At first he felt strongly inclined to vomit, but was quite unable to do so. He became alarmed, went to the nurse's room, and informed her of what had occurred. He was then attacked with violent purging, and severe vomiting. An emetic was given and vomiting was kept up by various means for nearly an hour. At the end of this time the man became faint; the surface was cold and pale; and the face and lips assumed a livid tint. The pulse was small, and almost imperceptible; and he was unable any longer to maintain the erect or sitting posture. Under treatment, the local pain and general distress were much decreased; the vomiting was arrested; and the surface of the body gradually regained warmth. On the fourth day the mucous membrane of the tongue and throat came away in shreds; and the uneasiness of the gullet was diminished. On the sixth day the patient had quite recovered, but felt rather weak. (Case by Dr. Adam, Ed. Med. Journ. 1855–6, vol. i, p. 932.)

The poisonous properties of croton oil are owing to a fatty acid (crotonic acid), which it contains in uncertain quantity. Probably this may explain why from six to ten drops of the oil may be sometimes given without causing much purging. It commonly begins to act speedily, i. e. within half an hour. The medicinal dose of it is from one to three drops. The oil acts as a poison on animals. Many instances of its effects on animals have been collected by Wibmer. (Arzneimittel. i, 215.) A singular case in reference to its effects on the horse, was the subject of a trial some years since. A veterinary surgeon administered, as a medicine, fifteen drops to a horse. The lips of the animal were swollen, and the skin peeled off; the horse suffered evidently great pain, and after lingering a short time, died. An action was brought by the owner of the horse at the Oxford Aut. Cir. 1838, for the recovery of its value. From the evidence then given, it seemed probable that the animal had really died from a small dose of the oil, although there was reason to believe that a larger quantity was given than was here alleged to have caused death. Wibmer mentions two instances in which twenty and thirty drops were given to horses without materially affecting them.

Analysis.—Croton *oil* is a fixed oil of a lemon-yellow color. It has a peculiarly unpleasant odor, and a hot acrid burning taste. It has a faint acid reaction, which it imparts to water; and as it is of lower specific gravity, it floats on the surface. It is very soluble in ether, and by this liquid it may be separated from organic liquids. When warmed with nitric acid, the oil is turned of a dark-brown color; and there is an abundant evolution of nitrous acid vapor.

Croton *seeds* are of an oval form, and about three-eighths of an inch in length. They are covered with a dusky thin bluish-colored brittle shell, having within a yellowish-white oleaginous and easily sectile kernel, which forms the great bulk of the seed. When boiled in a solution of potash holding dissolved some oxide of lead, they are blackened, thus indicating the presence of sulphur. Like all the varieties of vegeto-albumen, the kernel is turned of a deep red-brown color when it is boiled in concentrated muriatic acid.

COLCHICUM (MEADOW SAFFRON).

Meadow-saffron (COLCHICUM AUTUMNALE) contains a poisonous alkaloid,—colchicina,—the effects of which on animals are similar to those of veratria, the alkaloid existing in White hellebore. The most noxious parts of the plant are the bulbs (or roots) and seeds, but the leaves and flowers have also an irritant action.

Symptoms and Appearances.—The effects of colchicum are those of a vegetable irritant: it causes a burning pain in the gullet and stomach, with violent vomiting, and occasionally violent bilious purging, followed by death. The general nature of the symptoms may be gathered from five fatal cases, which occurred at the Toulon Hospital under the treatment of M. Roux. (L'Union Médicale, Mars 27, 1855; and Lancet, May 5, 1855, p. 474.) It appears that *two ounces* of colchicum *wine* were given to each patient by mistake in place of bark wine. None of the men experienced any ill effects until about *two hours* after they had taken the poison. Two of them, who had felt an unusual burning at the stomach with colicky pains, then began to vomit; and when examined, they were pale, cold, with a small pulse, and suffering from severe abdominal pain, nausea, constant vomiting, and frequent and abundant purging. In about six hours after the colchicum wine had been taken, there was a burning sensation in the throat and along the gullet, ardent thirst, and frequent yellowish serous evacuations, without mucus or blood. The mental faculties were unimpaired, and no alteration had taken place in the motor or sensory power. At five o'clock in the afternoon, nine hours after the colchicum had been taken, the symptoms were unchanged, except in one patient; in this case vomiting and purging had ceased, and the skin had become warm and moist. Three of the patients died after *nineteen hours'* suffering, and two after *twenty-six hours.* The last two were, shortly before dissolution, in a deplorable state, presenting lividity of the lips and nails, icy skin, and heaviness about the head; they complained, besides, of a vesical and rectal tenesmus, great thirst, burning of the throat, and severe pains in the loins and limbs. The *appearances* were similar in the five patients, and were as follows: No ulceration or traces of inflammation in the throat and gullet; stomach and intestines distended with a little gas, but containing a great deal of opaque fluid; mucous membrane much softened and red, but presenting no ulcerations; liver considerably congested, and spleen gorged with blood. No other alteration existed, except a strongly marked redness of the brain and spinal marrow. The aspect of the muscular tissue was rosy in all parts, and three days after death decomposition had not begun. No colchicina was found in the vomited matters, but by comparative analyses, it was proved that the men had been poisoned by colchicum.

In November, 1839, a gentleman swallowed by mistake one ounce and a half of *wine* of colchicum. He was immediately seized with severe pain in the abdomen; other symptoms of irritation came on, and he died in seven hours. No examination of the body was made! In another instance in which an ounce of the wine was taken, death occurred in thirty-nine hours. (Schneider's Annalen, i, 232.) In one case in which this dose was taken, the person recovered after suffering from cramps in the limbs and twitching of the tendons. (L'Union Méd. Aug. 24, 1848.) A woman, æt. 56, suffering from rheumatism, for whom wine of colchicum had been prescribed, took by mistake an ounce of the wine of the seeds, in divided doses, in twelve hours. She suffered from nausea, violent and profuse vomiting, slight purging, with heat and burning pain in the throat, great thirst, cold clammy skin, feeble pulse, pain in the stomach, and pain in the forehead. Inflammation of the stomach supervened, and the retching, vomiting, thirst, and pain continued for three days. She then recovered. (Amer. Jour. Med. Sci. Jan. 1857; and Brit. and For. Rev. vol. xix, 1857, p. 409.) In a well-marked case of poisoning by the *wine* of colchicum, reported by Mr. Fereday, two ounces were taken. The symptoms did not come on for an hour and a half; there was then copious vomiting of a yellow fluid, severe pain with great tenderness in the abdomen, tenesmus, and thirst. The patient died in forty-eight hours without being convulsed or manifesting any sign of cerebral disturbance. The chief morbid appearance was a patch of redness in the mucous membrane of the stomach, near the cardiac orifice; the intestines were slightly inflamed. The head was not examined. (Medical Gazette, x, 161. See also Casper, Ger. Med. p. 450.) A case of poisoning by the medicinal administration of colchicum has been communicated to me by Mr. Mann of Bartholomew Close. Three and a half drachms of the wine of colchicum were taken in divided doses, and caused death on the fourth day. There was no inflammation of the mucous membrane, but simply extravasation of blood into the mucous follicles.

Dr. Ollivier has published the details of two cases, in each of which about five ounces of the *tincture* of colchicum root were swallowed, and proved fatal. In one case there was continued and violent vomiting, but no purging: the pupils were not dilated; pulse thready and slow; intense thirst; severe cramps in the soles of the feet; intellect unaffected; no convulsions or tetanic spasms. The patient died in twenty-two hours. The body was not inspected until putrefaction had advanced to a degree to destroy all the appearances. An unsuccessful attempt was made to extract colchicina from the contents of the stomach. In the other case symptoms speedily appeared, indicated by violent pain in the abdomen; frequent vomiting but no purging; difficult breathing; pupils not dilated; coldness of the surface; no tetanic spasms, but cramps in the soles of the feet; pulse small; intellectual faculties preserved. Death took place in twenty-eight hours. The vessels of the pia mater were much injected, but there was no redness of the mucous membrane of the stomach. (Annales d'Hyg. 1836, ii, 394.)

The mucous membrane has been found softened in two cases of poisoning by the tincture. In two other cases in which an ounce and a half of the *tincture* was taken, and death ensued in forty-eight hours, no morbid appearances were found. (Casper, Ger. Med. 1857, p. 451.)

Seeds.—A case is quoted in the Pharmaceutical Times (Jan. 23, 1847, p. 354), in which a man, æt. 75, swallowed a large quantity of colchicum *seeds*. He soon experienced a burning sensation in the throat, with nausea, vomiting, violent colicky pains, and frequent purging. These were succeeded by difficulty of breathing, and discharge of bloody urine. After death, patches of inflammation and mortification were found in the stomach and small intestines (duodenum). The latter contained some colchicum seeds. A man, æt. 52, took a *decoction* made with a tablespoonful of colchicum seeds to a pint and a half of water. He was seized with vomiting and purging, continuing incessantly until death, which took place in about thirty-six hours. The only appearance of note was that the stomach had a violet or purple hue. Two cases of death from the fresh seeds are reported in the Journal de Chimie Méd. 1853, p. 421. The roots, seeds, leaves, and flowers of colchicum are poisonous. The reader will find a summary of the action of this plant in a paper by Dr. Maclagan. (Ed. Month. Journal, Dec. 1851.)

The medicinal doses of the vinegar and wine of colchicum are from half a drachm to a drachm; of the tincture from twenty to thirty minims, and of the powder from two to eight grains. According to Dr. Aldridge, the tincture, given frequently in medicinal doses, produces salivation. (Dub. Hosp. Gaz. Oct. 1845, p. 52.)

Analysis.—Colchicum owes its noxious properties to the alkaloid COLCHICINA, which exist both in the seeds and root. It is in fine white crystals, which have a bitter acrid taste. It is soluble in water, has a feeble alkaline reaction, and forms crystallizable salts with acids. In most of its reactions it resembles the other alkaloids. Its solutions have a bitter taste, give a white precipitate with tannic acid,—a yellow with chloride of platina, and a puce-brown with solution of iodine. Its special character is, that concentrated nitric acid produces with it a violet color, which changes to blue and brown.

Colchicina is a powerful poison. One-tenth of a grain killed a cat: vomiting, purging, and salivation were among the symptoms,—these were followed by convulsions. The stomach and intestines were highly inflamed, and blood was effused throughout their course. (Pereira, Mat. Med. vol. ii, pt. 1, p. 157.) In two cases less than half a grain proved fatal to adults. (Casper, Ger. Med. 1857, i, p. 402.) In order to extract this alkaloid, the contents of the stomach evaporated at a low temperature should be treated with alcohol acidulated with a few drops of hydrochloric acid. The alcoholic liquor should be evaporated at 100° to a syrupy consistency,—the residue treated with water, and filtered. The aqueous solution concentrated may be mixed with calcined magnesia, and then diluted with ether or chloroform. Either of these liquids will dissolve out the colchicina which may be obtained on evaporation.

BLACK AND WHITE HELLEBORE.

There are several species of Hellebore; but the two plants which are most commonly used as poisons under this name, are the Black and White Hellebore.

Black Hellebore.—This is the Helleborus Niger of the modern, and the Melampodium of the old pharmacopœias: it is also known under the name of *Christmas Rose*, from its flowering in January. In Lancashire it is called *Brank-ursine*. Another species, Helleborus Fœtidus, sparingly grows in shady places and on a chalky soil, flowering in March and April: it is known under the name of *Bear's foot*, *Setter-wort*, or *Helleboraster*.

Symptoms and Appearances.—According to Wibmer, the roots of the Black Hellebore possess the greatest activity: but the leaves are also highly poisonous when used in the form of infusion. By long boiling the poisonous properties of the plant are diminished, probably owing to the loss of the volatile principle, which is an acrid oil. The roots and leaves have a local irritant action, producing violent vomiting and purging in small doses, with severe pain in the abdomen, followed by cold sweats, convulsions, insensibility, and death. The powdered root, in a dose of a few grains, acts like a drastic purgative. In a case reported by Morgagni, half a drachm of the aqueous extract killed a man, aged 50, in eight hours. The symptoms were severe pain in the abdomen and violent vomiting. After death, the whole of the alimentary canal was found inflamed, but especially the large intestines. (Wibmer, op. cit. Helleborus.) A case is quoted by the same writer, in which a tablespoonful of the finely-powdered root (taken by mistake for rhubarb) caused severe symptoms of irritant poisoning, which did not disappear for four hours. The man recovered on the fourth day. The experiments performed by Orfila on animals show that this poison acts like a *local* irritant when applied to a wound on the skin. (Op. cit. ii, 369.)

Hellebore is a favorite remedy for worms with quacks and rural doctresses. It is not, therefore, surprising that it should be occasionally administered in an overdose, and cause death.

In the following case, which occurred to Dr. Massey, a woman recovered from a large dose of the infusion. About one ounce and a half of the root of the black hellebore was put in a covered jar in an oven with twelve ounces of water; it was allowed to remain a whole night by a slow fire. On the following morning the woman took a teacupful of the infusion. It produced pain and pricking in the tongue, fauces, and throat; to use her own expression, "as if a hundred pins were pricking her." There was a painful sense of constriction of the throat, with great difficulty of swallowing; pain at the stomach, and violent sickness. The tongue began to swell, as well as the parts about the throat, and much viscid mucus was voided from the mouth. The eyes were sunk; there was excessive prostration of strength, discoloration about the eyelids, with great collapse of the vital powers,—much as is seen in the collapse of Asiatic cholera; the extremities were cold, and the general surface of the body was bedewed with a cold, clammy sweat. The pulse varied from thirty to fifty beats in a minute, was very small, and at times scarcely perceptible. An emetic of sulphate of zinc was given with large quantities of lukewarm water; and this was followed by three grains of camphor dissolved in spirit of wine, and mixed with yolk of egg. The skin was kept warm by hot applications. Coffee was repeatedly given; and in the course of three or four hours the patient rallied considerably. The pulse rose to 68 and 70. She complained of headache; the bowels were relieved with castor oil; and beyond saline effervescing draughts nothing further was required. She soon recovered. (Lancet, July 26, 1856, p. 100.)

Mr. Todd, Coroner for Hants, forwarded to me the report of an inquiry which took place before him, in Nov. 1845, in which a child under two years of age was poisoned with an *infusion* of hellebore, administered to it by its grandmother, for the purpose of destroying worms. The leaves of the plant (Bear's foot) were bruised, and boiling water poured over them. Two dessert-spoonfuls were given to the child, who had been suffering from ague, but from which he had recently recovered. Within ten minutes after taking the mixture he was very sick, and was violently purged. The matter vomited was of a green color, and slimy: the sickness and purging continued until the evening, when he died, *i. e.* about thirteen hours after having taken the mixture. There were convulsions before death. On inspection, the whole body appeared blanched; the eyes were sunk, and the pupils dilated. There was diffused inflammation of the mucous membrane of the stomach, and a well-marked patch of inflammatory redness, about the size of a five-shilling piece, near its centre. The small intestines, which contained a brownish-yellow fluid, were much inflamed. The cæcum contained about thirty worms. The head and chest were not examined. Death was very properly attributed by the medical witness to the action of hellebore. The woman who prepared the infusion stated that she had frequently given it in large quantities to children, and there were no injurious effects. It is nevertheless to be regarded as an active poison; and if persons are not always killed by such worm-medicines, it must be regarded as a very fortunate circumstance. This acrid vegetable never can be given by an ignorant person without great risk.

Analysis.—This is confined to the botanical characters of the leaves and roots. Black Hellebore has a large flower with five round spreading petals, which are at first white, and afterwards become reddish-colored, and finally greenish. The flower of Fœtid Hellebore, or Bear's foot, has five oval concave petals, of a green color, tinged with purple at the margin.

White Hellebore. Veratrum Album.—The action of this plant is analogous to that of black hellebore; it is, however, more irritant. The powdered root produces a strong local effect, and causes violent sneezing. When taken internally, it gives rise to severe pain in the abdomen, violent vomiting and purging, followed by giddiness, dilatation of the pupils, convulsions, insensibility, and death. It produces a sense of great heat and constriction in the throat. In three cases mentioned by Dr. Pereira, in which the infusion had been swallowed, there was no purging. (Op. cit. vol. ii, pt. 1, p. 170.) A man swallowed by mistake half an ounce of white hellebore in powder. Four hours after taking the poison he was seen by a medical man. The chief symptoms were, a burning sensation in the mouth and throat, pain in the stomach, purging, nausea, but no vomiting until after an emetic had been given. There was no symptoms affecting the brain. Under treatment the man soon recovered. (Mr. Giles, in Lancet, 1857, vol. ii, p. 9.)

There can be no doubt that white hellebore is an irritant poison. The numerous observations collected by Wibmer prove that it acts powerfully on the system. In one instance, twenty grains of the powder caused convulsions and death in three hours, and in another, a man after eating the root died in six hours. Death was preceded by vomiting of bloody mucus, and by cold sweats. (Op. cit. Veratrum.) The smallest quantity required to destroy life is unknown. Dr. Christison quotes a case from Bernt, in which a man took but a small quantity of the powder, and died in the course of the day. After death, the same marks of irritation were found in the alimentary canal as those which have been described in treating of black hellebore.

Analysis.—Powdered *white hellebore* root has a reddish-brown color, resembling jalap. Nitric acid gives to it a red, rapidly passing to a dark-brown color. Sulphuric acid produces with it a dark-brown tint, almost black. Iodine water, a bluish-gray tint, slowly brought out. The proto and persalts of iron have no effect upon it.

Veratria.—White hellebore owes its noxious properties to the alkaloid *veratria*, which is itself a powerful poison. The late Mr. Callaway communicated to me the following fact. A physician prescribed medicinally for a lady, one grain of veratria divided into fifty pills, and three were directed to be taken for a dose. Not long after the dose had been swallowed, the patient was found insensible, the surface cold, the pulse failing, and there was every symptom of approaching dissolution. She remained some hours in a doubtful condition, but ultimately recovered. Supposing the medicine to have been well mixed, and the pills equally divided, not more than one-sixteenth of a grain of veratria was here taken! This case proves that pure veratria is capable of exerting a powerful effect. The common veratria of the shops is sometimes given medicinally, in doses of one-sixth of a grain. In the pure state, it forms a brownish-white uncrystalline powder, scarcely soluble in water even on boiling; but it is more readily dissolved by alcohol and ether. It has a faint alkaline reaction, and easily combines with acetic and other acids, forming soluble salts. It has a hot, acrid taste, without any bitterness. Strong nitric acid gives to it a light red, turning to an ochreous color. Diluted sulphuric acid, when heated with the powder, produces an intense crimson-red color. Veratria differs from colchicina in not being very soluble in water,—in not being crystallizable,—and in its power of exciting the most violent fits of convulsive sneezing.

CANTHARIDES—SPANISH FLIES.

Symptoms.—This animal poison has been frequently administered either in the state of powder or tincture, for the purpose of exciting aphrodisiac propensities, or of procuring abortion. When taken in the form of powder, and in the dose of one or two drachms, it gives rise to the following symptoms: a burning sensation in the throat, with great difficulty of swallowing; violent pain in the abdomen, with nausea and vomiting of bloody mucus; there is also great thirst, with dryness of the throat; but in a few cases observed by Mr. Maxwell salivation was a prominent symptom. As the case proceeds, pain is commonly experienced in the loins, and there is an incessant desire to void urine; but only a small quantity of blood or bloody urine is passed at each effort. M. Lavallée found that one effect of the poison, externally applied, was to give a strong albuminous character to the urine. (L'Union Médicale, 17 Juin, 1847, p. 308.) The abdominal pain becomes of the most violent griping kind. Purging supervenes, but this is not always observed: the matters discharged from the bowels are mixed with blood and mucus. In these, as well as in the vomited liquids, shining green particles may be commonly seen, whereby the nature of the poison taken is at once indicated. After a time, there is often severe priapism, and the genital organs are swollen and inflamed both in the male and female. In one instance, observed by Dr. Pereira, abortion was induced, probably owing to the excitement of the uterus, from the severe affection of the bladder: for there is no proof that this substance acts directly on the uterus to induce abortion.

With respect to the aphrodisiac propensities caused by cantharides, these can seldom be excited in either sex, except when the substance is administered in a quantity which would seriously endanger life. When the case proves fatal, death is usually preceded by fainting, giddiness, and convulsions. The *tincture* of cantharides produces similar symptoms: they are, however, more speedily induced, and the constriction and burning sensation in the throat and stomach are more strongly marked: these symptoms are often so severe as to render it impossible for the person to swallow, and the act of swallowing gives rise to severe pain in the throat and abdomen. The following well-marked case of poisoning by the *tincture* occurred to Dr. Ives, of New York. A boy, æt. 17, swallowed an ounce. When seen, an hour and a half afterwards, the respiration was hurried, there was profuse salivation, convulsive trembling, acute pain in the regions of the stomach and bladder, and such exquisite sensibility that the slightest pressure produced convulsions. These came in paroxysms, were accompanied by painful priapism, and followed by delirium. On the seventh day he was seized with pain in the head, trembling and universal spasms: coma followed. He then appeared to improve; but on the fourteenth day violent convulsions supervened, and these were followed by insensibility and death. (Beck's Med. Jur. 5th Ed. 842.) A woman swallowed a similar quantity of the *tincture*. Some time afterwards she suffered from severe pain in the abdomen, increased by pressure: it became swollen and tympanitic. She passed during the night a pint and a half of urine unmixed with blood. In two days, the pulse was feeble and scarcely perceptible: there was delirium, with severe pain in the region of the kidneys and bladder: the urine was continually drawn off by a catheter. It was more than a fortnight before she was convalescent. (Med. Gaz. xxix, 63.) A man swallowed sixty grains of the *powder* of cantharides by mistake for jalap. Some hours afterwards, the patient was found laboring under incessant vomiting, intense thirst, with burning pain in the mouth, throat, and stomach, countenance anxious, tongue swollen and thickly coated, pulse 130, weak and tremulous; the matter vomited had a greenish color, and a peculiarly offensive odor. There were frequent and urgent calls to micturition, always preceded by severe pain at the point of the penis; and the passage of the urine was attended with severe scalding. The urine was turbid, and slightly tinged with blood. There was a dull heavy pain in the lumbar region, increased by pressure, and occasional priapism. Vomiting was promoted, and a large quantity of a thick solution of gum arabic was administered at intervals. The patient rapidly recovered; his recovery was probably due to the greater part of the poison having been ejected by the early occurrence of vomiting. (Dr. Fisher in Med. Gaz. xxxix, 855.)

Cantharides are sometimes taken in the form of a blistering plaster. A case was reported to the Westminster Medical Society, in which a woman took a piece about the size of a walnut, in chocolate, by mistake. In about an hour, vomiting and strangury supervened: this was followed by inflammation of the kidneys. The woman speedily recovered. In another instance, in which half an ounce of the plaster, containing two drachms of the powder, was taken by a lunatic, æt. 45, death took place in twenty-four hours, although remedies were immediately applied. In about two hours the whole of the mucous membrane of the mouth was reddened, and covered with small blisters. In seven hours there was great coldness of the surface, with imperceptible pulse. The urine passed, was mixed with blood. (Ed. Med. and Sur. Jour. October, 1844, p. 563.) A singular case, in which an attempt was made to poison a man by blistering plaster, was recently the subject of a trial in France. This person perceived, after taking some soup, a strong and bitter taste, for which he could not account. He also suffered from violent pain in the stomach and abdomen, especially in the region of the bladder; and he could only, under scalding pain, void a small quantity of urine, tinged with blood. He recovered from these symptoms; but three months subsequently, and two hours after taking some soup which had the same bitter taste, they returned in an aggravated form. They were relieved by doses of olive oil and milk to excite vomiting. A few days afterwards, he found in this soup a dark-colored substance, which, on examination by a medical man, turned out to be cantharides. His brother-in-law, who was proved to have recently purchased blistering plaster, was tried on a charge of attempting to poison him. One-half of the plaster sold to the prisoner was found, and it was proved to contain about thirty-one grains of powdered cantharides. The medical witnesses agreed that the symptoms, under which the prosecutor had labored, were those which commonly result from this poison; but one of them contended that the dose administered was not sufficient to cause death! (The exact quantity taken is not stated.) The accused was nevertheless capitally convicted. (Journal de Chimie, 1846, p. 606.)

Chronic poisoning.—It is not often that we have occasion to observe poisoning by cantharides in a chronic form; but a remarkable set of cases has been reported by Mr. Frestel, which show that, contrary to common belief, the substance does not invariably excite those aphrodisiac propensities which have been generally ascribed to it. It appears that six young men (students) had, during a period of six months, unknowingly taken with their food powdered cantharides by mistake for pepper. The quantity taken was at no time large, but very variable. The only marked general symptom was, great restlessness. There was no affection of the nervous system, nor any disorder of the bowels. The appetite was unaffected. No pain was experienced in the renal or lumbar regions. About three hours after the meal, there was a slight pruritus of the glans, with a desire to micturate, and there was also *ardor urinæ.* The desire for micturition continued for from two to four hours, and then gradually ceased, leaving some irritation about the urethra. There was neither priapism nor any erotic feeling. The absence of symptoms is the more remarkable, as the substance must have been taken in very different doses at different times. Without knowing the cause of the disorder from which they had suffered, they employed for their relief, warm baths, and an abundance of warm demulcent drinks. This plan of treatment was found to be most effectual. (Journal de Chimie Médicale, Janvier, 1847, p. 17.)

Effects of external application.—It is proper to state that cantharides will operate as a poison when applied externally to a wound, or ulcerated surface, or even when applied to a large surface of healthy skin. In January, 1841, a girl, æt. 16, was killed at Windsor under the following circumstances. She was affected with the itch: sulphur ointment was prescribed for her; but, by mistake, blistering ointment was used. This was rubbed all over the body of the girl:—she was soon seized with violent burning pain,—the ointment was immediately washed off, but the cuticle came off with it. The girl died in five days, having suffered from all the usual symptoms of poisoning by cantharides.

Appearances after death.—In one well-marked case of poisoning by cantharides, the whole of the alimentary canal from the mouth downwards, was in a state of inflammation, as well as the ureters, kidneys, and internal organs of generation. The mouth and tongue seemed to be deprived of their mucous membrane. In Dr. Ives's case, above mentioned (p. 511), in which an ounce of the tincture was swallowed, and death did not occur for fourteen days, the mucous membrane of the stomach was not inflamed; but it was pulpy and easily detached. The kidneys were, however, inflamed. The brain has been found congested, and ulceration of the bladder is said to have been met with. There are very few fatal cases reported, in which the appearances have been accurately noted; indeed, the greater number of those who have taken this poison, have recovered. In the case of the lunatic (supra)—on an inspection of the body, the vessels of the brain were found gorged with blood, and a quantity of serum was effused in the ventricles and between the convolutions. The heart and lungs were healthy. The internal surface of the stomach was covered with red points interspersed with ecchymosis, in the centre of which was seen an adhering particle of the powder of cantharides. The intestines were healthy, but the kidneys were red and gorged with blood. The left ureter internally was of a very red color. The bladder was thickened, and the mucous coat injected with blood. (Ed. M. and S. J. Oct. 1844, p. 563.) In a case which occurred to Mr. Saunders, death took place in about twenty-four hours. The deceased must have taken the greater part of half an ounce of cantharides in powder. The symptoms were such as have been above described. On inspection the vessels of the brain were filled with dark-colored blood, and the ventricles were distended with serum. Both lungs were highly engorged with dark-colored blood. The gullet was partially inflamed, and there were patches of inflammation on the mucous coat of the stomach, which had become detached in several places. The same inflammatory appearance existed in the small intestines, in the folds of which the powder of cantharides was abundantly seen. The vessels were distended, and the liver was engorged with dark blood. The gall-bladder was much distended with bile, and none of this secretion appeared to have passed into the bowels. The spleen and kidneys were highly congested; the ureters were inflamed; the bladder contracted and empty, and its internal surface pale. The glittering of the particles of cantharides in the viscera during the inspection by candlelight was very remarkable. (Medical Times, Feb. 3, 1849, p. 287.) Cantharides are sometimes described as a corrosive poison; but the substance appears to have no local action of a chemical nature. It is a pure *irritant*, and the effects observed are entirely due to irritation and inflammation. Serious accidents have frequently occurred from the powder of cantharides having been mistaken for jalap, cubebs, and other medicinal substances. A man lost his life on one occasion, by having cantharides-powder supplied to him for cubebs, in a druggist's shop.

Fatal dose.—This has been a frequent subject of medico-legal inquiry. The medicinal dose of the powder is generally stated to be from one to two grains. In a case which was the subject of a criminal investigation, a medical witness stated, that one grain was the maximum dose; but according to the late Dr. Thomson it may be increased to *three* grains. The dose of the London Pharmacopœial tincture is from ten minims gradually increased to one fluid drachm,—of the powder, from *one to two grains*. (Pereira, Mat. Med. ii, pt. 2, p. 754.) Doses above this, whether of the powder or the tincture, are likely to be injurious, and to give rise to symptoms of poisoning. On a trial which took place at Aberdeen, in 1825, it appeared that a drachm of the powder had been administered: severe symptoms followed, but the person recovered. Dr. Dyce, the medical witness, said he had given ten grains of the powder at a dose as a medicine. In three cases, observed by Mr. Maxwell, a drachm of the powder mixed with six ounces of rum was taken by each person; they were robust, healthy negroes,—they suffered severely, but recovered in about ten days:—in these cases, irritation of the urinary organs did not appear until after the men had been bled. The *smallest quantity* of powder which has been known to destroy life, was in the case of a young female, quoted by Orfila; the quantity taken was estimated at *twenty-four grains* in two doses. She died in four days; but as abortion preceded death, it is difficult to say how far this may have been concerned in accelerating that event. Her intellect was clear until the last. In one instance a man recovered from a dose of fifteen grains of the powder. (Canstatt's Jahresbericht, 1844, v, p. 301.) In another, the patient recovered after having taken a dose of twenty grains. (Ed. Med. and Surg. Journal, Oct. 1844.) In a third, which occurred to Dr. Fisher, there was recovery after a dose of sixty grains (ante, p. 512), and in a fourth after a dose of two drachms or one hundred and twenty grains. (Med. Gaz. vol. xlii, p. 873.)

An *ounce* of the tincture has been known to destroy life in fourteen days. This, I believe, is the smallest fatal dose yet known. (See Dr. Ives's case, p. 511.) Four drachms and even six drachms of the tincture have been taken; and although the usual symptoms have followed, the patients recovered. A case of poisoning by cantharides was the subject of a trial at the Central Criminal Court, in September, 1836. Six drachms of the tincture were administered to a girl, æt. 17; the medical witness was required to say whether half an ounce was sufficient to kill, as also what proportion of cantharides was contained in the tincture. One ounce of the tincture, P. L., is equivalent to six grains of the powder; but considering that the principle *Cantharidine* is the substance on which the poisonous properties depend, it is very likely that the tincture may vary in strength according to its mode of preparation. A case is quoted by Pereira, from Dr. Hosack (Mat. Med. ii, pt. 2, p. 750), in which it is stated that six ounces of the tincture were taken by a man without causing dangerous symptoms! This must have been an extraordinarily weak preparation; and probably the insects from which the tincture was made contained little or no cantharidine. The same writer mentions a case within his own knowledge, in which one ounce of the tincture caused serious symptoms. In the annual registration returns for 1840, only one case of death from cantharides is recorded in a man, æt. 46.

Treatment.—When vomiting exists, this may be promoted by warm demulcent liquids, as thick linseed-tea, or a strong solution of gum arabic: if it does not exist, emetics and castor-oil should be given,—the object being to dislodge the poison. Demulcent injections may also be used. The inflamed state of the throat may not admit of the application of the stomach-pump. Oil was formerly regarded as an antidote; but it has been found that this is a ready solvent of the active principle, and it is therefore injurious.

Analysis.—*Cantharidine* is the vesicating and, at the same time, the poisonous principle of the insect. It is a white solid crystallizable substance, insoluble in water; but soluble in ether, chloroform, alcohol, fixed oils, and caustic alkalies. Although water does not dissolve it in its pure state, it takes it up with other principles from the powdered insect; and thus an infusion of cantharides is poisonous. Chloroform will separate it from its watery solution. It is not volatile according to Mr. Proctor under 220°. It is powerfully irritant, but requires solution in some menstruum and contact with the skin to produce irritant effects. There are no chemical characters by which this principle can be safely identified, if we except its *vesicating* properties. Orfila has applied reagents to detect cantharidine in the tincture; but without success. It has been recommended to digest the suspected solid, or the liquid contents of the stomach evaporated to an extract, in successive quantities of ether,—to concentrate these ethereal solutions by slow evaporation, and then observe whether the concentrated liquid produces vesication or not: the medical jurist being expected, in such cases, to make himself the subject of experiment. In this way, Barruel discovered cantharides in some chocolate. (Ann. d'Hyg. 1835, i, p. 455.) This mode of testing is somewhat uncertain, unless the quantity of poison is large; and the affirmative evidence which it yields is better than the negative; since we can hardly infer the absence of the poison when we obtain no result. There is, however, no other mode of discovering cantharides in solution, whether as tincture or infusion, than this. In this process, chloroform may be substituted for ether. The difficulty of extracting *Cantharidine* may be conceived, when it is stated that, according to Thierry's experiments, which are the most perfect, the quantity contained in the fly is only about the 250th part of its weight, so that it would require nearly half an ounce of the powder, to yield one grain of cantharidine. The quantity of cantharidine required to produce vesication is extremely small. Robiquet found that the 1-100th part of a grain, placed on a slip of paper and applied to the edge of the lower lip, caused small blisters in a quarter of an hour.

Cantharides are most commonly taken in powder, and then we may easily recognize the poison by its physical characters. If the insect be entire, or only coarsely powdered, there can be no doubt of its nature. However finely reduced, the powder is observed to present, by reflected light, small golden-green or copper-colored scales. These are perceptible to the eye, and are very distinct under a common lens. It has been recommended to separate the particles of cantharides, by suspending the liquid or other contents of the stomach in warm water, when the insoluble powder will subside, and the particles may be collected and dried for examination. In an elaborate essay on this subject (Ann. d'Hyg. Oct. 1842), M. Poumet recommends that the suspected liquid, mixed with rectified spirit, should be spread on sheets of glass, and the liquid allowed to evaporate spontaneously to dryness. The green and copper-colored scales may then be seen, on examining by reflected light either one or both surfaces of the glass. He also found that the particles adhering to the mucous membrane of the stomach or intestines, may be easily detected by inflating the viscus, and allowing it to become dry in the distended state, taking care to attach to it a heavy weight, so that during the process of drying, all the folds of the mucous membrane may disappear. On cutting the dried membrane and opening it on a flat surface, the colored scales become perceptible. Physical evidence of this kind would not be of much avail for medico-legal purposes, unless there were concomitant evidence from symptoms and appearances. On trials for administering this poison, the analysis might be confined to a portion of the article administered; and the physical test is then applicable, since the powder is commonly given in large quantity and adheres closely to the mucous membrane. There are, however, many insects, besides cantharides, which have wings of a golden-green color, and are not poisonous: yet such insects are not likely to be found in the form of powder in the human stomach. M. Poumet states that there are some cantharides which contain no cantharidine. Particles of cantharides may be detected in the viscera long after interment. Orfila has detected them after a period of nine months, so that they do not seem to be affected by the decomposition of the body.

The evidence of the presence of cantharides, or of their having been taken, is necessary to support a criminal charge; for, however unambiguous the symptoms produced by this poison may appear to be in its peculiar effects on the generative and urinary apparatus, a medical jurist should be aware that similar symptoms may proceed from disease. An important case of this kind occurred to Dr. Hastings. (Med. Gaz. xii, 481.) A young lady was suddenly seized with vomiting, thirst, pain in the loins, strangury, and considerable discharge of blood from the urethra: the generative organs were swollen and painful. She died in four days. She was governess in a family, and there was some suspicion that she had been poisoned by cantharides. The stomach, kidneys, and bladder were found inflamed: the latter organ contained about two ounces of blood. There was no trace of poison, and indeed it was pretty certain, from the general evidence, that none could have been taken.

PHARMACEUTICAL PREPARATIONS.—The *doses* and *comparative strength* of the powder and tincture of Cantharides, according to the London Pharmacopœia, have been already stated. There are some other preparations, the strength of which it may be important for a medical jurist to know. The ACETUM CANTHARIDIS or Vinegar of Cantharides is used externally. It is equivalent to about one-tenth of the powder; *i. e.* five ounces are equal to four drachms of powdered cantharides. It contains eight times as much cantharides as the tincture. The CERATUM CANTHARIDIS contains one-sixth, and the EMPLASTRUM CANTHARIDIS contains one-half of its weight of the powder.

Among other irritants of an organic nature may be mentioned, Aloes, Anemone, Arum, Bryony, Capsicum, Celandine, Colocynth, Daffodil, Elaterium, Elder, Euphorbium, Gamboge, Hedge Hyssop, Physic nut, Jalap, Manchineel, Mezereon, Mustard, Ranunculus, Scammony, Sorrel, Stavesacre, Castor-seeds, Creasote, Oil of Tar, Oil of Turpentine, Pyroxylic spirit, Dippel's oil, Fusel oil, Decayed vegetable food, Poisoned grain, Ergot of rye, Poisonous fish and other kinds of poisonous animal food.

NEUROTIC POISONS.

CEREBRAL OR NARCOTIC POISONS.

CHAPTER XXXIII.

GENERAL REMARKS ON NEUROTIC POISONS—OPIUM AND LAUDANUM—SYMPTOMS—PERIOD OF COMMENCEMENT—DEATH AFTER REMISSION—CHRONIC POISONING—OPIUM-EATING—EFFECTS OF EXTERNAL APPLICATION—APPEARANCES AFTER DEATH—FATAL DOSE—DEATH FROM SMALL, AND RECOVERY FROM LARGE DOSES—FATAL EFFECTS ON INFANTS—PERIOD AT WHICH DEATH TAKES PLACE—TREATMENT.

THE effects produced by the Neurotic class of poisons on the system have been already described (page 105). They are chiefly referable to disorder of the brain, spinal marrow, and nervous system. The most prominent symptoms are headache, giddiness, paralysis, insensibility, and convulsions. The brain is the organ upon which a cerebral or narcotic poison primarily acts; but in some cases, by the occurrence of convulsions, there is an indication of a remote effect on the spinal marrow. The distinction between irritant and neurotic poisons is well marked, so far as symptoms are concerned. Neurotic poisons are destitute of any acrid or corrosive properties: they have no local chemical action on the mouth and fauces, and they rarely give rise to vomiting or purging. When they prove fatal, they do not commonly leave any well-marked appearances in the stomach or bowels like the irritants. There is sometimes a fulness of the vessels of the brain and its membranes; but effusion of blood is rarely observed. It is usually said that they do not produce any redness of the mucous membrane of the stomach or intestines: this appearance, has, however, been met with on several occasions in poisoning with Prussic acid. Opium does not cause inflammation of these organs, and when this condition has been found, it may probably be ascribed to the action of alcohol, in which the opium has been dissolved.

The first group of Neurotic poisons (p. 103) includes those substances which primarily and specially affect the brain. Of these Opium and Prussic acid may be taken as types.

OPIUM—LAUDANUM.

General remarks.—OPIUM is a solid vegetable extract—the concrete juice of the unripe capsules of the *Papaver somniferum*. It is sometimes taken in this state as a poison, but more commonly in solution in alcohol under the form of tincture, or as it is popularly called—LAUDANUM. Its poisonous properties are principally due to the presence of an alkali, *Morphia*, which exists in it in a state of combination with a vegetable acid, the *Meconic*. Opium contains a proportion of morphia—varying from two per cent. in the Bengal variety to about nine per cent. in certain varieties obtained from the East Indies. According to some chemists, good opium will yield from ten to thirteen per cent. of morphia. The Turkey opium contains on an average about six per cent. according to the analysis of Mulder: but the best kinds of Smyrna opium contain thirteen per cent. (Pereira, Mat. Med. vol. ii, p. ii, p. 606.) This difference in the quantity of morphia contained in the drug, may sometimes account for certain differences observed in the effects produced by particular doses.

There is a difference of opinion respecting the strength of *laudanum* or the pharmacopœial tincture of opium. According to some, one fluid drachm, or sixty minims of the tincture, is equivalent to *five grains* of opium, while the London Pharmacopœia assigns only about *three grains*. The strength of the tincture as it is procured of different druggists varies greatly. (See Pereira, Mat. Med. vol. ii, pt. ii, p. 647; also Lancet, March 12, 1853, p. 251; and Pharm. Journal, 1851, p. 250.)

There is no form of poisoning so frequent as that by opium and its various preparations. In two years, there were one hundred and ninety-six fatal cases in England and Wales (p. 219), forming nearly two-thirds of all the cases of poisoning. One-seventh of these were cases of children poisoned by overdoses of opium or its compounds, and most of the others were the result of suicide or accident. It is calculated that *three-fourths* of all the deaths from opium take place among children *under five years of age!* This, however, forms but a small proportion of the actual number of cases; since there is no kind of poisoning wherein recoveries are so frequent.

The poisonous salt of opium, meconate of morphia, is soluble in water, alcohol, and diluted acids. The aqueous and alcoholic solutions have an acid reaction. The solutions have a peculiar taste and odor, and by the latter the presence of opium may be in general recognized. The taste is bitter.

Extract of opium may be regarded as a pure form of the drug. It contains a larger proportion of the poisonous alkaloid morphia. Three grains of extract are equal to about five grains of crude opium. It is employed in medicine in doses of from one quarter of a grain to three or four grains. The alcoholic solution, under the name of *laudanum*, is sold to the public, in quantities of from half a drachm to two drachms, for twopence,—from two drachms to four drachms, for fourpence,—exceeding this quantity, eight pence and one shilling per ounce. It is very often sold by ignorant drug-dealers for tincture of rhubarb or black draught.

SYMPTOMS.—The symptoms which manifest themselves when a large dose of opium or of laudanum has been taken, are of a very uniform character. They consist in giddiness, drowsiness, a strong tendency to sleep, stupor, succeeded by perfect insensibility, the person lying motionless, with the eyes closed as if in a sound sleep. In this stage he may be easily roused by a loud noise, and made to answer a question; but he speedily relapses into stupor. In a later stage, when coma has supervened with stertorous breathing, it will be difficult, if not impossible, to rouse him. The pulse is at first small, quick, and irregular, the respiration hurried: but when the individual becomes comatose, the breathing is slow and stertorous: the pulse slow and full. The skin is occasionally cold and pallid,—sometimes livid: at other times warm and bathed in perspiration. The pupils are sometimes contracted, at others dilated. From cases which I have been able to collect, contraction of the pupils is much more frequent than dilatation. In a case referred to me, in 1846, one pupil was contracted and the other dilated. They are commonly insensible to light. The expression of the countenance is placid, pale, and ghastly: the eyes are heavy, and the lips are livid. Sometimes there is vomiting, or even purging; and if vomiting take place freely before stupor sets in, there is great hope of recovery. This symptom is chiefly observed when a large dose of opium has been taken; and it may be perhaps ascribed to the mechanical effect of the poison on the stomach. The peculiar odor of opium is occasionally perceptible in the breath. The muscles of the limbs feel flabby and relaxed, the lower jaw drops, the pulse is feeble and scarcely perceptible, the sphincters are in a state of relaxation, the temperature of the body is low, there is a loud mucous rattle in breathing, and convulsions are sometimes observed before death; these are more commonly met with in children than in adults. One of the marked effects of this poison is to suspend all the secretions except that of the skin. During the lethargic state. the skin, although cold, is often copiously bathed in perspiration. It is a question yet to be determined, whether this may not be the medium by which the poison is principally eliminated. Nausea and vomiting, with headache, loss of appetite, and lassitude, often follow on recovery.

With a view of settling some disputed questions respecting the symptoms produced by opium in a healthy person, Dr. Böcker, in the year 1849, induced a man, æt. 50, to submit to a series of experiments. The doses of opium given to this man were gradually increased, until confirmed symptoms of poisoning showed themselves. The man soon recovered from the influence of the drug. The following results were obtained: The first effects were those of incipient drunkenness,—depression,—weakness of the muscles,—trembling of the limbs, especially of the knees,—dizziness,—weight in the head,—giddiness,—stupor, followed by profound sleep,—nausea,—eructations, with vomiting,—cramps,—convulsions,—contraction of the sphincters of the rectum and bladder,—contraction of the pupil,—rarely purging, but in general obstinate constipation. The pulse, at first frequent, became gradually slower and normal, but weak. The breathing was at first free, but afterwards more difficult; and the skin was moist. Under still larger doses of opium, there were hallucinations, with delirium, and symptoms of congestion of the brain, attended at first with excitement, and subsequently with depression. At first the skin was cool; but afterwards there was itching. In a more advanced stage, the face appeared either swollen and livid, or pale. A deathlike sleep seized the person,—the pupils were dilated,—there was trembling of the limbs,—paralysis, especially of one side,—anæsthesia (loss of sensibility),—the pulse scarcely perceptible,—great difficulty of breathing, with symptoms of asphyxia, or apoplexy. In rare cases, trismus and tetanus appear. Death, generally speaking, rapidly follows a relaxed state of the sphincters, and paralysis of the muscles. (Die Vergiftungen, 1857, p. 50.) This record of the effects of opium is of value, as it is the result of actual observation. It tends to reconcile the discrepant statements of others in reference to certain symptoms,—*e. g.* the contracted or dilated state of the pupils, the dilated state being confined to the advanced stage of poisoning from large doses. At the same time, it is to be observed, that the above series of symptoms were the result of progressively increased doses. In practice, a large dose is taken at once; and the first rapidly passes into the second stage, without the intermediate conditions described by Böcker.

The symptoms usually *commence* in from *half an hour* to *an hour* after the poison has been swallowed. Sometimes they come on in a few minutes, especially in children; and at others their appearance is protracted for a long period. In a case reported by Dr. Skae, the person was found totally insensible in *fifteen minutes.* As we might expect, from the facts connected with the absorption of poisons, when the drug is taken in a *solid* state, the symptoms are commonly more slow in appearing, than when it is *dissolved* in alcohol. Their appearance is also protracted if the stomach be full at the time: and it is said that intoxication has the effect of retarding them. In a case reported by Mr. Semple (May, 1841), a dose of one ounce and a half of laudanum was taken by a girl, æt. 19: the symptoms did not appear until *an hour and a half* afterwards. There was drowsiness, but the patient was rational; the pupils were contracted to the size of a pin's head, and did not dilate on removing the light. Under treatment the girl recovered. In a case in which I was consulted, a girl died in about twelve hours from the effects of a large dose of opium, and there was some reason to believe that the confirmed symptoms of narcotic poisoning did not show themselves until *three hours* after she had swallowed the poison. (Med. Gaz. vol. xxxvii, 724.) With the exception of an extraordinary instance mentioned by Dr. Christison, the longest period to which the symptoms have been protracted was *five hours.* This occurred in a case reported by Dr. Sewell. A man, æt. 40, swallowed ten drachms of tincture of opium by mistake for tincture of rhubarb. When seen by Dr. Sewell, five hours afterwards, he was in bed, awake, and quite conscious. The pupils were contracted to the size of a pin's point, and immovable; the temporal arteries pulsated with great violence; he spoke with difficulty, the skin was dry, the pulse 100 and jerking. By active treatment, the man recovered in about fifteen hours. (Dublin Hosp. Gaz. Oct. 15th, 1845, p. 78.) In Dr. Christison's case, a man swallowed an ounce and a half of laudanum, and in an hour, half as much more, and lay down in bed. Some excitement followed, with numbness of the extremities. He continued sensible, and so lively for *seven* hours after the first dose, that a medical man did not believe his statement. It was not until the *eighteenth* hour that stupor set in, and in two hours more the symptoms of [illegible] nin[illegible] y opium were of an aggravated kind. He finally reco- [illegible] under [illegible] atment, but there was no apparent cause for this extreme [illegible] he symptoms. (Op. cit. 706.) In the case which occurred [illegible] *ine hours* elapsed before the usual symptoms were mani- f[illegible] et, July 25th, 1857.)

[illegible] ually anomalous nature of the symptoms and appearances in [illegible] ni[illegible] opium is well shown in a case communicated to me in November, [illegible] 0, by Mr. Clegg. A man, æt. 56, in good health, swallowed by mistake in two pills from twenty-eight to thirty grains of opium of commerce. This was at three o'clock in the morning, and he died rather suddenly at one o'clock in the day, *i. e. ten hours* after taking the drug. In about an hour after he had taken it, he was heard to moan; there was twitch[illegible], and copious perspiration, with alteration of spee[illegible] s senses, was a little drowsy at intervals, and vo[illegible] ccasionally. [illegible] dressed himself as usual in the morning, and con[illegible] d of sev[illegible] in his stomach. He was seen by a medical man [illegible] o hou[illegible] his death: there was no coma, contraction of the [illegible] ymptom of poisoning by opium. Even the vomited [illegible] m [illegible] f the drug. He walked in a trembling way. A [illegible] l that he could have swallowed opium, no emetic was given.

The period at which the cerebral [illegible] toms commence is a question of some importance in relation to the [illegible] tion of power on the part of a person, to perform certain acts ind[illegible] of volition and locomotion after having swallowed a large dose of t[illegible] ison. Thus the narcotic effects may not come on until the deceased has had ample time to attempt suicide in some other way. In March, 1843, a gentleman committed suicide at Hammersmith: he was found suspended by a silk handkerchief; but it was shown that he had previously swallowed a large dose of laudanum. There was no doubt that he had died from hanging. In general, it must be allowed as at least possible that a person who has taken a sufficient quantity of this poison to prove fatal, may move about and perform many acts for one or two hours afterwards, but this power ceases when the cerebral symptoms commence. In a fatal case of poisoning by opium which occurred to Dr. Skae, he ascertained that the person rose from his bed, and had moved about his room, at least two and probably three hours after having taken the poison. (Ed. Med. and Surg. Journal, July, 1840.) In another instance, in which the quantity taken was probably half an ounce, but enough to destroy life, the person was able to converse cheerfully and readily with a neighbor two hours after she had swallowed the poison.

It has been frequently observed, in cases of poisoning by this drug, that a person has recovered from the first symptoms, and has then had a relapse, and died. There is some medico-legal interest connected with this state, which has been called secondary asphyxia from opium, although there appears to be no good reason for giving to it this name. In December, 1843, a gentleman swallowed a quantity of laudanum, and was found laboring under the usual symptoms. The greater part of the poison was removed from the stomach by the pump; and he so far recovered from his insensibility as to be able to enter into conversation with his medical attendant; but a relapse took place, and he died the following night. It is not improbable that, in these cases, death may be occasioned by the accumulation of the poison, carried by the absorbents into the system; *i. e.* the morphia may be more rapidly carried into the system than it is eliminated out of it. A remarkable case illustrative of this *remittent* form of poisoning by opium has been published by Mr. Kirby. (Dubl. Med. Press, Dec. 24, 1845, 406.) A lady, æt. 30, swallowed six dessert-spoonfuls of the common tincture of opium, having previously taken four glasses of port wine, and some spirits in her tea. After three hours she was found in her bed, perfectly insensible. The skin was pale and cold; the limbs were relaxed, and felt flabby when handled. She breathed slowly, and apparently with great difficulty. There was a loud mucous rattle in the windpipe, with great frothing at the mouth, which was wide open. The eyelids were closed, and, when raised by the finger, exhibited a distorted, fixed, suffused eyeball, with an extremely contracted pupil; the pulse was 100, and very feeble. The temperature of the body was low. There was paralysis of the muscles of the throat, with intense coma. From this state she recovered so as to recognize her medical attendant and her domestics. In about eighteen hours from the time of taking the poison there was a relapse, indicated by great difficulty of breathing. She referred all her sufferings to her "lungs," asked for a blister for her back, and announced that she was dying. The wrist was now pulseless, and there was coldness and relaxation of the limbs. She died in twenty-three hours after she swallowed the laudanum, retaining her consciousness until a few moments before her death. Dr. Christison quotes a case from Pyl, in which, after a large dose of opium, there was a complete remission of symptoms; but paralysis came on, and the man died in ten days. (Op. cit. 710.) He thinks that opium could only act in this manner by calling forth some disposition to natural disease—some organic affection of the brain being suddenly developed through the cerebral congestion occasioned by the poison. In reference to Pyl's protracted case, this may be the true explanation; but, in the other instances cited, it appears to me we can only refer the fatal symptoms, notwithstanding the remission, to the direct effect of the poison. After all, at an inquest, the practical question would be reduced to this: Would the deceased have died under the same circumstances had he not taken the dose of opium? Whether the drug acted directly to destroy life, or indirectly by inducing some fatal disease, it must equally be regarded as the immediate cause of death. It would be a great mistake, however, to assume that because there had been a remission or intermission of symptoms, the drug could not have caused death.

The case of the late Mr. A. Stafford (November, 1857) is in this respect of some importance. This gentleman was laboring under severe pain in the abdomen, as it was supposed from gall-stones, and, to relieve this pain, he was bled to thirty ounces, and took, by medical prescription, in the space of four hours, about 200 drops of laudanum and 200 drops of tincture of henbane! This was equivalent to about 16 grains of opium, given within a short period to a person unaccustomed to the drug. The usual noxious effects were produced, and the most severe treatment by flagellation on the soles of the feet was resorted to for many hours, in order to prevent the patient from falling into a fatal sleep. In the course of six days, although much debilitated, he had so far recovered as to be able to make a journey to Dublin for further medical advice. He was then in a state of great debility, but he had recovered from the primary effects of the narcotic medicines. He died on the fifth day after his arrival, and on the eleventh day from the treatment. On inspection, the lungs were found healthy, but there were about five ounces of serum in the pleural cavities. The heart was larger than natural, very soft, its parieties were thin and in a state of fatty degeneration. It floated in water. No particular appearances were found in the abdomen, if we except the presence of many biliary calculi in the gall-bladder: there were none in the duct. The stomach contained a little fluid, but, with the intestines, was normal. In the head there was sub-arachnoid effusion, but no congestion of the brain or membranes, and no serum in the ventricles. There was a slight fulness of the vessels at the base. The cause of death was ascribed to a diseased condition of the heart. Assuming the correctness of this view, the administration of such large doses of opium, with the loss of a considerable quantity of blood, must, as a general rule, place a person in great jeopardy. This was not one of those cases of tolerance which would justify the use of such large doses of opium. In fact, the narcotized state in which the deceased remained for at least twelve hours, proved that the medicine had passed from its medicinal to its poisonous operation.

It is proper to remember that a person may die from opium without being necessarily narcotized up to the time of death. (See cases, pp. 522, 523.) The narcotic symptoms may disappear, and be replaced by others affecting the lungs (case, p. 523), which may be just as fatal to the patient. The term "opiatized" would be more appropriate than "narcotized," to indicate the noxious effects on these exceptional occasions.

Chronic poisoning by opium. Opium-eating.—When opium is taken for a long period in small doses, which are gradually increased, its effects are very different. It is this state which we witness in those persons who are addicted to opium-eating. There is no poison which appears to be so much under the influence of habit as opium (p. 91). Böcker met with several opium-eaters,—one of them a physician, who took daily thirty grains of solid opium. The English opium-eater (De Quincy) took at one time, for a daily quantity, nine ounces of laudanum. The injurious effects of the drug thus taken for a long period of time, have already given rise to an important question in law relative to life-insurance (case of the Earl of Mar, 1828); and it will be, therefore, proper to state those facts which have been ascertained with respect to the influence of this practice on the health. One of the best descriptions of the effects of opium-eating is that given by Dr. Oppenheim in his account of the state of medicine in Turkey. He says, "The habitual opium-eater is readily recognized by his appearance. A total attenuation of body,—a withered, yellow countenance,—a lame gait,—a bending of the spine, frequently to such a degree as to cause the body to assume a circular form,—and glassy, deep-sunken eyes,—betray him at the first glance.

The digestive organs are in the highest degree disturbed; the sufferer eats scarcely anything, and has hardly one evacuation in a week; his mental and bodily powers are destroyed. As the habit becomes more confirmed, his strength continues decreasing, the craving for the drug becomes greater; and, in order to produce the desired effect, the dose must be constantly augmented. After long indulgence, the opium-eater is subject to neuralgic pains, to which opium itself brings no relief. These persons seldom attain the age of forty if they have begun to use opium early." This description of the effects is exactly what we should expect from physiological and pathological reasoning. Dr. Christison states, that he has ascertained that constipation is by no means a general consequence of the continued use of opium; but there may be exceptions to the rule. It is believed by some that the action of the drug is different in different countries, and that this description of the effects produced by the use of opium in Turkey is not applicable to the English opium-eater. There is no reason, however, for entertaining this opinion. The late Dr. A. T. Thomson noticed, in reference to one of his patients who had been in the habit of taking opium, that if she passed over the usual time for taking a dose, she felt the most distressing sensations about the joints, not of pain, but such as she was unable to describe. She suffered from involuntary motions of the arms, fingers, and toes; numbness in the limbs and body generally; profuse perspiration, nausea, vomiting, and loss of appetite; a saline taste in the saliva, and a bad taste in the mouth; a general impairment of the senses; trembling of the limbs, with a sense of great debility and lassitude. The mental powers were also impaired, and this state was attended by a miserable depression of the spirits. These symptoms were relieved by a repetition of the dose. One of the effects of opium in this case was to produce constipation. A remarkable instance of chronic poisoning by opium, which occurred to Dr. Myers, will be found in the Edinburgh Med. Jour. 1855–6, vol. i, p. 357.

The chronic form of poisoning, as witnessed among children in the factory districts, has been described by Mr. Grainger. It appears that laudanum, and other preparations of opium, are given to children in gradually increased doses, until the child will bear from fifteen to twenty drops of laudanum at a time. The child becomes pale and wan, with a peculiar sharpness of feature, and rapidly wastes away. The majority of these children die by the time they are two years old.

Effects of external application.—Opium, and all preparations containing morphia, have the property of affecting the body through the skin. Excepting in cases of idiosyncrasy, or where a large quantity of the drug is too frequently applied to an abraded surface, they are not likely to produce fatal effects by this mode of introduction into the system. There is, however, an instance reported of a very large quantity of laudanum having acted fatally when applied in a poultice to the unbroken skin of the abdomen. (Christison, op. cit. 723.) A soldier, æt. 32, was attacked with phlegmonous erysipelas on the face and outer part of the right leg, on account of which a linseed poultice, moistened with laudanum, was applied to the limb. Next morning he was found in a state of deep sopor: the eyelids tremulous and half open, pupils contracted, the lips distorted, the muscles of the face affected with spasm, and those of the limbs with convulsions. The medical attendant, perceiving a strong odor of opium, examined the bandages, and found them soaked with laudanum, the hospital servant having ignorantly applied nearly an ounce! In spite of treatment the convulsions increased, the pulse became more feeble, and the patient died. On inspection, some red points were seen on the arachnoid: a strong opiate odor was exhaled from all parts of the body, and the heart, stomach, and brain were healthy. None of the poison could be detected in the blood. (Ed. M. and S. J. xxix, 450; and Journal de Chimie Médicale, Avril, 1827.) The application of opium, in any form, to an abraded surface of skin, is liable to give rise to all the results of narcotic poisoning.

In general, the narcotic preparation is only applied after the skin has been removed by a blister: but the following case is sufficient to show that small doses of a salt of morphia may in this way act very energetically. A young woman, laboring under scirrhus of the uterus, and suffering from vomiting and pain in the stomach, was ordered to apply to the pit of the stomach, from which the skin had been previously removed by a blister, the 1-23d part of a grain of the muriate of morphia. The same dose was repeated by the endermic process the following morning. Some time afterwards, the woman fell into a state of complete narcotism. She suffered from pain in the head, stupor, ringing in the ears, dizziness, and incoherency, a hot and dry skin, and a strong and frequent pulse. Among the symptoms was one somewhat remarkable, namely, that she saw only the half of surrounding objects: for instance, in the case of a person standing before her, she could only see the right or left half of the body. The cerebral congestion was followed by convulsions. Venesection was performed; but this only produced a stronger attack, followed by another. A compress, soaked in vinegar, with ice, was applied to the forehead, followed by mustard poultices to the lower extremities. The symptoms gradually abated, but it was three weeks before vision and speech were perfectly restored. (Oesterreichische Medicinische Wochenschrift, April, 1845.) Four-tenths of a grain of the acetate of morphia applied to a blistered surface have been known to cause dimness of vision and delirium. The dose commonly recommended for application endermically, is from one to two grains.

Opiate preparations introduced into a wound, or as enemata into the *rectum*, may also produce fatal effects. Orfila relates the case of a man who died from the effects of an injection containing thirty grains of opium. (Op. cit. ii, 225.) A child has been killed by ten grains of the sulphate of morphia, given in the form of an enema, by mistake for sulphate of quinine. (Med. Gaz. iv, 220.) In the case of Major Forester (Legal Examiner, October, 1852), a question arose respecting the relative effects of opium administered by the *mouth* or *rectum*. An injection had been ordered for this gentleman containing two drachms of *sedative solution* of opium, with half a drachm of camphorated tincture of opium. The whole of this was used as an injection, and the deceased took, in addition, from eight to fifteen grains of Dover's powder. In about an hour and a half he fell into a state of stupor, and died shortly afterwards, clearly from narcotic poisoning. Before death, he was seen by a medical man, who found him asleep, without stertorous breathing,—the skin perspiring,—and the pupils half dilated, but contracting perfectly to light. Assuming that the sedative solution used had the strength of tincture of opium, the dose was not unusually large, as,—according to English authorities (Christison, Op. cit. p. 723)—opium acts less powerfully by the rectum than by the mouth. In fact, it is asserted that twice as much is required to produce the same effect by injection as by the mouth. The deceased probably took, in this case, within a short period, at least seven grains of opium; and this quantity would account for his death by whichever channel it had been introduced. It has been elsewhere stated, that some poisons act more energetically and speedily by the rectum than by the mouth (p. 37); but this is, I believe, not in accordance with English experience regarding opium. I have known five grains, used as a suppository with soap, produce drowsiness and narcotism. This quantity would have probably caused death if it had been taken by the mouth (see p. 529).

The application of any opiate preparation to the lining membrane of the nose, will produce all the usual symptoms of poisoning and death. (Wibmer, Op. cit. Papaver.)

APPEARANCES AFTER DEATH.—In a case of poisoning by opium, which proved fatal in fifteen hours, examined at Guy's Hospital, the vessels of the head were found unusually congested throughout. On the surface of the anterior part of the left hemisphere there was ecchymosis, apparently produced by the effusion of a few drops of blood. There were numerous bloody points on the cut surface of the brain: there was no serum collected in the ventricles. The stomach was quite healthy. Fluidity of the blood is mentioned as a common appearance in poisoning by opium. There is also engorgement of the lungs; most frequently, according to Dr. Christison, in those cases which have been preceded by convulsions. (Op. cit. 732.) Among the external appearances, there is often great lividity of the skin. Extravasation of blood on the brain is rarely seen; serous effusion in the ventricles, or between the membranes, is much more common. The stomach is so seldom found otherwise than in a healthy state, that the redness, said to have been occasionally met with, may be regarded as probably due to congestion from accidental causes. In a case referred to me, in August, 1844, in which a woman, æt. 56, died in twenty-two hours after taking half an ounce of tincture of opium, the following appearances were found twenty-four hours after death. The brain was congested throughout, the blood in the sinuses fluid, and there was about an ounce of serum effused between the membranes and in the ventricles. The lungs were emphysematous in places, and the liver was rather large and congested. The intestines, nearly empty, had patches of congestion, with bloody mucus on the lining membrane. The stomach presented at the larger end some redness of the mucous membrane, but apparently of a pseudo-morbid character. The contents consisted of a dark, pasty, offensive mass, of a greenish color, intermixed with lumps of half-digested animal food: there was no smell of opium to be perceived, nor could the least trace of meconic acid or morphia be detected in them by the most careful analysis.

In a case of poisoning by a large dose of tincture of opium, Dr. Sharkey found the following appearances twelve hours after death. The body was warm and rigid; the stomach healthy, containing a quantity of a gruel-like fluid, without any smell of opium. The intestinal canal and the viscera of the abdomen were healthy. The veins of the scalp, as well as of the dura mater and sinuses, were gorged with blood; but there was no effusion in any part of the brain. The contents of the stomach yielded no trace of morphia or meconic acid; but there was no doubt that death had been caused by opium, taken the previous night. (Med. Gaz. xxxvii, 235.) In another case of death from a small dose of extract of opium, this gentleman found great lividity of the skin of the neck. The scalp, superficial veins, and sinuses of the brain, contained a large quantity of blood, both fluid and coagulated, around the upper part of the spinal marrow. There were patches of ecchymosis on the mucous membrane of the stomach. The heart was flaccid, pale, and nearly empty. The large veins, and the venous system generally, were much gorged. The case of an infant of six months, which was the subject of a trial at the Aberdeen Autumn Circuit, 1853, was communicated to me by Dr. Ogston. The child died in a few hours from a dose of sixty drops of the wine of opium. The only marked appearance in the body was congestion of the brain. In a case which I had to investigate a few years since, a child, aged fourteen months, was killed in eighteen hours from the effects of a dose of infusion of opium, equivalent to from three to five grains of the powder. The inspection of the body was made about twenty-four hours after death. Externally, there were a few livid spots on the skin of the abdomen, back, and thighs. The eyelids were open, and the eyes sunk in the orbits. The pupils appeared contracted, but the condition of the iris was not particularly noticed during life. The viscera of the chest were healthy; there was no mark of effusion or of any organic disease. The right cavities of the heart were congested. The viscera of the abdomen were also healthy, except the kidneys, the structure of which had undergone some change from disease. The stomach was healthy; the mucous membrane presented no trace of inflammation or disease in any part. It contained about a teaspoonful of a white viscid liquid, apparently consisting of milk and mucus in a semi-digested state. There was no farinaceous or any other food present, and no smell of opium; nor was the slightest trace of morphia or meconic acid detected in it on analysis, although the child had not vomited, but had remained throughout in a state of insensibility. The intestines were healthy. The bloodvessels of the brain were found much congested; but there was no effusion or extravasation of blood or serum.

According to Böcker, the usual appearance in opium-poisoning are,—great congestion of the sinuses and membranes of the brain,—an increased effusion of cerebro-spinal fluid,—effusion of serum in the ventricles of the brain, and in the subarachnoid tissue. The brain itself is often pale and bloodless; but sometimes there is effusion of blood in the ventricles, or in the subarachnoid cells. In the chest the lungs are much congested, and there are effusions of blood in the pulmonary tissue. The heart is filled with a large quantity of dark-colored blood. The stomach and bowels are generally normal,—rarely reddened in the mucous membrane;

but the liver, spleen, and kidneys are greatly congested. The urinary bladder is distended with urine,—a fact, which has led to the erroneous notion, that the secretion of urine is increased as a result of the action of the poison. It simply arises, however, from a loss of power of expulsion, as a result of paralysis and of a loss of consciousness in the person laboring under the effects of the poison.

In Mr. Clegg's case (ante, p. 522), there was no fulness of the vessels of the brain, or of its membranes; but there was such congestion at the greater end of the stomach, as to lead to the supposition that irritant poison had been taken. No irritant poison was found; but there were distinct indications of opium. The poison appears in this case to have produced no effect on the brain, but to have exerted its influence chiefly on the nerves connected with speech and motion. In Mr. Kirby's case (ante, p. 523), of death after a remission and relapse under severe pulmonary symptoms, the body was inspected after nineteen hours, and was then warm and rigid. The limbs were strongly extended, and the hands clenched. The whole front was pale, while all the underlying parts were deeply ecchymosed. The lungs filled the chest, and scarcely retired on exposure; they crepidated when handled, and yet they felt firm and fleshy, and resisted the scalpel. Their anterior surface presented a bloodless appearance, studded with small black spots of a circular form. Internally, they were gorged with livid blood. The large pulmonary vessels were nearly empty, as was the heart, the blood which they contained being fluid. The stomach was distended by a brownish-colored inodorous fluid. The mucous membrane exhibited purple marks from congestion. The intestines were pale throughout, except near the gall-bladder, where they were stained with bile. The voluntary muscles were very florid. The head was not examined.

From this account of the appearances, it will be seen that there is nothing but a fulness of the vessels of the brain, and greater or less congestion of the lungs, which can be looked upon as indicative of poisoning by opium; and even these are not always present. This condition of the brain or lungs, however, if it exist, can furnish no evidence of poisoning, when taken alone, since it is so frequently found, as a result of morbid causes, in the bodies of persons who have died from disease.

Fatal Dose.—The medicinal *dose* of opium, in *extract* or *powder*, for a healthy adult, varies from half a grain to two grains. Five grains would be a very full dose. The medicinal dose of the *tincture* for an adult is from ten minims to one drachm,—as an average, from *thirty* to *forty minims*. In a case, which occurred in London, in 1838, a man, æt. 45, was killed by ten grains of solid opium; and in September, 1843, a woman, æt. 88, was killed by eight grains of the drug given in two doses. (Brit. and For. Med. Rev. Oct. 1844, 558.) In another instance, a lady, æt. 29, suffered from giddiness, numbness of the limbs, and other serious symptoms, when only *twenty minims* of the tincture were introduced in the form of enema, *i. e.* about one grain and a half of opium. The same dose had been administered for six nights previously, without any serious effects following;—this fact renders it not improbable that the drug possesses an *accumulative* power. She continued in a state of delirium for twenty hours, and the numbness of the limbs only ceased after forty-eight hours. This was an unusually small dose thus to affect an adult by the rectum (page 526). Dr. Babington has communicated to me the case of a lady who had taken five grains of Dover's powder, *i. e.* about *half a grain of opium*, and who suffered from stupor and drowsiness for three days. It is well known that some persons cannot bear the smallest doses of opium without suffering from the most intense headache, and other painful symptoms. Dr. Christison states that he was acquainted with a gentleman who, even when in perfect health, could not take seven drops of laudanum (one-third to one-half of a grain of opium) without being narcotized. A man who had been suffering from piles, was ordered a starch-clyster containing only *one grain* of pure opium. In about half an hour after its administration, the patient was found in a partial state of narcotism. There was pain in the head, which felt hot, the eyes were red and suffused, glassy-looking, and half open; the face flushed; the tongue dry; and there was a painful tenderness both in the head and face on the slightest motion. There was complete loss of power in the upper and lower extremities, and the man was in a half-stupefied condition. (Dr. Steinthal in Casper's Wochenschrift, Mai, 1845, p. 294.) These serious effects produced by small doses on adults must, however, be considered as exceptions to the rule: they appear to be due to idiosyncrasy, or to a peculiar susceptibility of the poisonous effects of opium in certain constitutions. It must not be forgotten, however, that they lead to one important inference in legal medicine, namely, that an adult may be killed by a dose of opium, which many, relying upon a limited experience, would pronounce to be innocuous. We have commonly no means of detecting or recognizing the existence of this idiosyncrasy in individuals prior to the fatal event.

In a case communicated by Dr. Brown to Dr. Christison, four grains and a half of opium, mixed with nine grains of camphor, killed a man in nine hours with all the symptoms of narcotic poisoning. The *smallest dose of solid opium* which has been known to prove fatal to an adult, was in a case reported by Dr. Sharkey, of Jersey. (Med. Gaz. xxxvii, 236.) A stout muscular man, æt. 32, swallowed two pills, containing each about one grain and a quarter of extract of opium,—a quantity equivalent to *four grains* of crude opium: he was soon afterwards attacked by a convulsive fit, and died. In the case of a sailor, three drachms of the tincture proved fatal, in spite of early treatment. (Dr. Gibb, Lancet, July 25, 1857.) The *smallest fatal dose of the tincture* in an adult, which I have found recorded, is *two drachms*. The case is reported by Dr. Skae. (Ed. Med. and Surg. Journ. July, 1840.) The patient was a robust man, æt. 56; he swallowed the tincture at ten in the evening, and died under the usual symptoms the following morning; the case thus lasting only twelve hours. The quantity actually swallowed, however, appears to be involved in some doubt; for it is subsequently stated (p. 160) that *half an ounce* of laudanum may have been taken. Opium, as meconate of morphia, was detected in the stomach. In a case which I was required to investigate, a woman died in twenty-two hours after taking *half an ounce* of tincture of opium by mistake for tincture of rhubarb.

Recovery from large doses.—In July, 1857, a woman was brought to

Guy's Hospital in a state of complete narcotism, with contracted pupils, relaxed muscles, and all the other symptoms of acute poisoning by opium. It was ascertained from her husband, who was an opium-eater, that she had taken a scruple (*twenty grains*) of solid opium. The stomach-pump and other remedial measures, including galvanism, were employed; she recovered, and left the hospital in a few days. Very large doses of the tincture have been frequently taken without proving fatal. Two women, who had each swallowed an ounce of the tincture, were placed under treatment at St. Thomas's Hospital, and they both recovered. (Med. Gaz. xix, 264.) Several similar cases have occurred at Guy's Hospital. In July, 1841, a man was admitted, who had swallowed one ounce and a half of the tincture. Vomiting came on, but he was not brought to the hospital until twelve hours after he had taken the poison. The stomach-pump was employed, and even under these disadvantageous circumstances he recovered. Neither the matter then vomited, nor the liquid brought off by the instrument, contained any trace of opium or of meconate of morphia, although the quantity examined amounted to six ounces. About the same time a woman was brought in, who had swallowed ten drachms of laudanum by mistake for tincture of rhubarb. Vomiting ensued, and she did well. It appears reasonable to attribute these recoveries from large doses to early vomiting, or to the treatment employed; but this explanation is not always applicable. A case occurred to Dr. Young (Med. Gaz. xiv, 655), in which a young lady took an ounce of laudanum in whiskey, and recovered in five days: there was no vomiting, and the cause of the symptoms was not even suspected until she had recovered from her stupor, and confessed that she had taken the poison. Another case occurred at the Westminster Hospital (Lancet, Dec. 1843), in which a woman, æt. 25, was brought into that institution while laboring under symptoms of poisoning by opium. She was perfectly comatose, the features were devoid of expression, the lips purple, and the pupils contracted to the size of a pin's head. The eyes were everted and fixed. Sulphate of zinc and tartar emetic were given without effect, and the stomach-pump was not brought into use until about an hour after her admission. The contents of the stomach were entirely free from the smell of opium. The woman was kept roused, coffee was administered, and she recovered. It was ascertained that she had swallowed one ounce of laudanum, but at what time before her admission, is not stated. In this case, a long time had elapsed before the contents were removed from the stomach, and then there was no trace of opium to be perceived by the smell. Two cases of recovery in young men are reported by Mr. Kirby, in which an ounce and a half of the tincture had been swallowed. They were seen immediately, and owed their recovery to energetic treatment. (Dub. Med. Press, Dec. 24, 1845, 407.) The following instance of recovery from a large dose of opium was privately communicated to me a few years since by one of my class. A medical student, after a hearty supper, at nine o'clock in the evening, swallowed *four ounces* of tincture of opium (P. L.), made by himself from opium procured at a respectable druggist's. He went to bed and slept until six o'clock the next morning, when he was awakened by a feeling of nausea. He then vomited freely; and, as he supposed, the whole of the contents of the stomach, smelling strongly of opium, were ejected. He perfectly recovered without feeling any other symptom than inability for muscular exertion. It is remarkable that the opium should have remained so many hours on the stomach without causing serious symptoms and death. I have elsewhere recorded a case in which *five ounces* of laudanum were taken without producing sleep, and the patient recovered. (Guy's Hosp. Reports, Oct. 1850, p. 220.) Dr. Gibb met with a case in which a man, æt. 72, swallowed an ounce and a half of laudanum. The poison remained in his stomach for *nine hours* without causing the usual symptoms. There was slight drowsiness, with a disposition to doze. At the end of this time he vomited the greater part of the laudanum, and recovered. (Lancet, July 25, 1857.)

Some remarkable results connected with the effect of *habit* on the fatal dose of opium, have been elsewhere detailed. (See ante, p. 91 and 524.) It is popularly known that opium-eaters will bear very large doses with impunity. It would take a dose of some ounces to kill such individuals. The Turks begin with from one to two or three grains, and increase the quantity gradually until it amounts to two, three, or in many instances six drachms. From three to eight ounces of laudanum are set down as the regular daily allowance of an English opium-eater. The following case is mentioned by Mr. Kirby. A gentleman, of a very haggard, sallow, gloomy appearance, and trembling all over, entered a druggist's shop and asked for an ounce of laudanum, which was properly refused without medical sanction. He then asked to be shown how much an ounce was, and when it stood before him, measured in a graduated glass, he seized it from the counter, and eagerly swallowed it. The druggist was struck powerless. The gentleman, seeing his great alarm, assured him it was needless, as he had been a long time an opium-eater, and took, twice daily, as much as he had just drunk. He remained for half an hour in conversation with the druggist, who was surprised to observe the wonderful changes which so short a period had made in his visitor, whose manner became animated, at the same time that his face became plump, and suffused with a pleasing blush. (Dub. Med. Press, Dec. 24, 1845, p. 407.)

Fatal effects of opium on infants.—In connection with this subject, it is important to bear in mind, that *infants* and young persons are liable to be killed by very *small doses of opium;* they appear to be peculiarly susceptible of the effects of this poison. Dr. Ramisch, of Prague, met with an instance of a child four months old, that was nearly killed by the administration of one grain of Dover's powder, containing only the tenth part of a grain of opium;—the child suffered from stupor and other alarming symptoms. The following case occurred in June, 1832. Four grains of Dover's powder (containing less than half a grain of opium) were given to a child four years and a half old. It soon became comatose, and died in seven hours. Dr. Kelso met with an instance in which a child, nine months old, was killed in nine hours by four drops of laudanum, equal to only *one-third part of a grain* of opium: it was much convulsed before death. A case is referred to in a late number of the Medical Gazette, in which two drops of laudanum, equal to the *sixth-part of a grain* of opium, killed an infant. The following is another illustration of the fatal effects of a similar dose. A nurse gave to an infant, five days old, *two drops* of laudanum, about three o'clock in the morning. Five hours afterwards the child was found by the medical attendant in a state of complete narcotism. It was revived by a cold bath, but a relapse came on, and it died the same evening, about eighteen hours after the poison had been given to it. On inspection, the brain and abdominal viscera were found in a perfectly healthy state, and there was no smell of opium in the stomach. (Prov. Med. Jour. Oct. 28, 1846, p. 519.) The fatal dose here, as in the former case, was equal to the sixth of a grain of opium, and to only an infinitesimal dose of morphia!

Another case is reported (Lancet, Feb. 1842) in which a child two days old, was killed by a dose of a mixture containing one minim and a half of tincture of opium, equal to the *eighth-part of a grain.* The child was seized with narcotism, followed by coma, and died in fourteen hours. There are no appearances in the body under these circumstances to indicate the cause of death, and it is not probable that the poison, when taken in so small a quantity, could be chemically detected by the odor or chemical tests. Dr. Pereira saw a case in which a powerful effect was produced upon an infant by one drop of the tincture of opium. (Op. cit. ii, pt. 2, p. 649.) Dr. Merriman met with an instance where a child a month old was thrown into excessive stupor by a single drop of the tincture (equal to the *twelfth-part of a grain* of opium), contained in a dose of mixture prescribed for it. He met with two instances in which death was caused by a small dose of Godfrey's cordial, which contains opium.

Among recent cases showing the fatality of this drug to infants, are the following. Dr. E. Smith attended a child which died comatose eighteen hours after having taken *one minim* of the tincture of opium (1-12th grain). Symptoms of narcotic poisoning set in, in about half an hour, and the child never rallied from the effects of the drug. (Med. Times and Gaz. April 15, 1854, p. 386.) Dr. Edwards, of Liverpool, communicated to me a case which occurred under his observation, in November, 1857, in which an infant under four weeks died from a dose of paregoric, equivalent to 1-90th part of a grain of opium. The child became comatose in half an hour, and remained in this state for seven hours, when it died, having been slightly convulsed before death. Another case is recorded, in which two drops of laudanum destroyed a child four days old. (See Provincial Journal, April 8, 1846, p. 168.)

On the other hand, infants have recovered from doses of a grain and upwards. Dr. O'Rorke describes a case in which a child of seven months recovered in eighteen hours from a dose of laudanum equivalent to between two and three grains of opium. (Med. Times and Gaz. May 15, 1858, p. 511, and July 24, p. 95.) A nurse gave to a new-born child thirty minims (= 2½ grains of opium) of tincture of opium. The effects produced were deep, hurried respiration,—great lividity of the skin, especially of the arms and legs,—closed eyes,—contracted pupils, —coma,—vomiting, and convulsions. Convulsive spasms occurred at irregular intervals, when the eyes stared, with twitching and contraction of the muscles of the face. The child did not vomit until a convulsive fit was about to come on. The matter vomited was not tested. The fits became gradually less severe, and finally ceased after some hours. The child quite recovered. (Lancet, Aug. 29, 1858, p. 220.) Probably the greater part of the draught was ejected. Mr. Murray met with a case in which three-fourths of a teaspoonful of laudanum, equivalent to three grains of moist opium, were given to an infant a year old. The usual symptoms followed: but under active treatment the child recovered in twenty-four hours. It was especially noticed that the cerebral symptoms were alleviated when there was copious perspiration. (Edinburgh Med. Journal, Feb. 1858, p. 718.)

In spite of such facts as those above related, there is often a disposition among medical witnesses to refer the death of a child in these cases to natural diseases, because the quantity of opium administered is small. As an illustration of this may be mentioned the following case. A woman gave a child four weeks old a narcotic draught, containing *an eighth of a grain* of opium and as much hyoscyamus. The child fell fast asleep, and died comatose in twelve hours. Contrary to well-known facts, the physician asserted that the child could not have been poisoned by so small a dose! A case was tried at the Chester Summer Assizes, 1847 (Reg. v. Deays), in which a question on this subject was put to a medical witness. He was asked whether *four drops* of tincture of opium would kill an infant, and properly replied in the affirmative. The mother was charged with the murder, but acquitted.

Dr. J. B. Beck published in the New York Journal of Medicine some useful remarks upon the effects of opium on infants. He shows that while this drug has a much greater influence on an infant than on an adult, in consequence of the greater impressibility of the nervous system, it is at the same time much more uncertain in its operation; and it is thus liable to prove fatal in very small doses. Among the instances which he has accumulated, illustrative of the powerful action of the drug, he mentions one in which a child was narcotized by twenty drops of paregoric-elixir, equal to the *twelfth part of a grain* of opium. This essay has been republished in the Medical Gazette for March, 1844 (vol. xxxiii, p. 767). A case is reported, in which, under judicious treatment and adverse circumstances, a child of nine months recovered after having swallowed twenty-five minims of laudanum (Dub. Med. Press, April, 1846.)

PERIOD AT WHICH DEATH TAKES PLACE.—It has been remarked that most cases of poisoning by opium prove fatal in from about six to twelve hours. Those who recover from the stupor, and survive longer than this period, generally do well. From some cases which have occurred, it would seem that there may be a partial recovery, and afterwards a fatal relapse. The symptoms, however, either progress steadily to a fatal termination, or the stupor suddenly disappears, vomiting ensues, and the person recovers. Several instances are recorded of this poison having destroyed life in from seven to nine hours. One has occurred within my knowledge in which an adult died in five hours after taking the drug prescribed for him by a quack. Dr. Christison met with a case which could not have lasted above five, and another is mentioned by him which lasted only three hours. Dr. Beck quotes a case which proved fatal in two hours and a half. (Beck, Med. Jur. 873.) I have notes of two cases in which death occurred in *two hours*. In one of these, communicated to me by Mr. Clegg (Sept. 1854), a man, æt. 29, took about an ounce of laudanum. He died within two hours, without any sign of convulsions. The other case occurred at Chatham (July, 1856): a man swallowed about three ounces of laudanum by mistake for senna. He died in two hours, after having suffered from the usual symptoms. The most rapid case of death yet reported was that of a soldier who was accidentally poisoned, in September, 1846, in the Hospital of Val de Grace. It appears that he swallowed by mistake about an ounce of laudanum, and died in convulsions in *three-quarters of an hour* afterwards. (Journal de Mêdecine, Octobre, 1846, p. 475. For a similar case, see Med. Gaz. vol. xlv, p. 743.) It is possible that the drug may kill even with greater rapidity than this; but as a medico-legal fact, we are at present entitled to state, that it has destroyed life within the short period above mentioned. On the other hand, the cases are sometimes much protracted. There are several instances of death in fifteen or seventeen hours. I have known one case fatal in twenty-two hours, and among those collected by Dr. Christison the longest lasted twenty-four hours. (Op. cit. 712.) In eight fatal cases of poisoning by opium reported by Dr. Beck, the smallest quantity taken was one drachm,—the largest, one ounce and a half. The shortest time between the taking of the poison and death was eight hours,—the longest, twenty hours,—the average time of six cases was fourteen hours. (Dub. Med. Press, May, 1845.) In the same journal it is stated, on Dr. Beck's authority, that out of thirty-nine fatal cases of poisoning by laudanum, the smallest quantity taken was one drop, the largest sixteen ounces. The shortest period for death was *two hours*, the longest *forty-eight hours;* average of twenty-three cases, twelve hours. (For a case fatal after fifty-six hours, see Med. Times and Gaz. March 20, 1858, p. 292.)

TREATMENT.—The first object is to remove the poison by the stomach-pump, or in the case of an infant, by a catheter, as speedily as possible. The instrument should be employed until the water used for washing out the organ has no longer the color or smell of opium. The entire absence of the drug may be perhaps better indicated by adding to the liquid a few drops of a solution of a persalt of iron. If no red color be produced, there is reason to believe that there is no meconic acid, and therefore no opium, present. In thus removing the poison, we at once arrest the progress of absorption. Emetics are of no service unless the individual possesses the power of swallowing. Occasional doses of sulphate of zinc may then be given to him, and in the intervals, a decoction of strong coffee or tea. Cold affusion on the head, chest, and spine, has been adopted with great success: in the treatment of infants the plunging of the body into a warm bath, and suddenly removing it from the water into the cold air, has been found a most effectual method of rousing them. (Med. Gaz. xxv. 878.) Flagellation to the palms of the hands and soles of the feet or the back, has also been successfully employed. A common plan for rousing an adult is to cause him to keep in continual motion, by making him walk between two assistants. Above all things, the tendency to fall into a state of lethargy must be prevented. If called to an adult or infant already in a lethargic condition, the application of shocks to the head and spine by an electro-magnetic apparatus will be found most effectual. It has in several instances led to recovery when the person was in an almost hopeless condition. An illustration of the effects of an overdose of this drug on a child, and of the benefit derivable from the electro-magnetic treatment, is furnished by a case, reported by Mr. Colahan. (Dub. Med. Press, April 22, 1846, 244.)

In a case in which two ounces and a half of laudanum had been taken, and the patient refused to submit to the usual treatment, an emetic was injected per anum, and it is reported, with success. (Med. Gaz. viii, 12.) Artificial inflation of the lungs has been employed in the lethargic state, but it is of doubtful benefit, since the great object is to apply a direct stimulus to the nervous system. Bleeding should not be resorted to until *after* the poison has been completely removed from the stomach, as the abstraction of blood acts injuriously by tending to promote absorption. It is, indeed, only justifiable when, during the second stage, there is a strong and full pulse, with symptoms indicative of cerebral congestion. If the pulse should become feeble, and sink after the abstraction of a small quantity of blood, the operation should be carried no farther. There are very few cases in which bleeding is necessary, and, as Mr. Bullock has remarked, its tendency is rather to weaken the patient and protract recovery. (Med. Gaz. xix, 264.) While all agree that copious bleeding is injurious, there is a great difference of opinion as to the propriety of bleeding in any case. A loss of blood tends to increase the debility under which the person may be laboring, and thus to accelerate death. This question was of material importance in the case of the late Mr. A. Stafford (p. 523). The deceased, while suffering from the effects of a powerful dose of opium, was bled to thirty ounces. In addition to this, there was subsequently a further loss of blood from the accidental slipping of the bandages. The medical opinion at the inquest was, that the bleeding was injurious. (See report of the case, Med. Times and Gazette, Dec. 12, 1857, p. 600.) When there are signs of recovery, ammonia applied on rags to the nostrils, and frictions to the chest with the compound camphor liniment, will aid in restoring the patient. Poultices of mustard and cayenne pepper have been applied with advantage. The means above stated, variously employed, have been found eminently successful; but especially the removal of the poison by the stomach-pump. Out of numerous cases of poisoning by opium, brought to Guy's Hospital, but very few have proved fatal, even when the remedial treatment was applied late. Vinegar was formerly recommended, but it is known to be a powerful solvent of morphia and other poisonous alkaloids. Its use, therefore, has long been abandoned as decidedly objectionable. A case of poisoning by laudanum is reported, in the treatment of which vinegar was given in two-ounce doses every half hour. The effect was to increase the stupor, and other bad symptoms. (Med. Gaz. xxxviii, 683.) Orfila has recommended tannic acid in the form of galls, oak bark, cinchona, or very strong black tea, as a chemical antidote, because this acid has the property of precipitating morphia from its solution in the form of a comparatively insoluble tannate. If this substance has acted beneficially, it has most probably been by its stimulant and astringent properties. At any rate, as the tannate of morphia is not more insoluble than pure morphia itself, and it is perfectly soluble in the acid secretions of the stomach, it is difficult to admit Orfila's chemical explanation.

A strong decoction of coffee has been frequently employed as a stimulant to promote recovery, and apparently with benefit. Böcker affirms that in cases in which there is a tendency to congestion of the brain and lungs, this liquid operates injuriously; and that there is no case recorded in which recovery can be assigned, in any shape, to the use of coffee. (Vergiftungen, 1857, p. 144; Ann. de Therapeutique, 1847, p. 303.) It appears to have been given on the principle that it causes wakefulness, while opium produces sleep. On the same grounds he condemns the use of brandy and camphor. The stimulating plan of treatment is one which demands further inquiry.

M. Bouchardat has recommended the employment as a chemical antidote of the ioduretted iodide of potassium. According to him, this solution forms insoluble and inert compounds with the alkaloids, especially with morphia. The product, he states, is even insoluble in weak acids. The proportions which he recommends are, iodine three grains, iodide of potassium six grains, water one pint. It is recommended to be given in doses of a wine-glassful, while vomiting should at the same time be promoted by emetics. (Gazette Médicale, Janvier 9, 1847, p. 31.)

CHAPTER XXXIV.

POISONING BY POPPIES—GODFREY'S CORDIAL—DALBY'S CARMINATIVE—PAREGORIC ELIXIR—DOVER'S POWDER—OPIUM LOZENGES—BLACK DROP—SEDATIVE SOLUTION—POISONING BY THE SALTS OF MORPHIA—SYMPTOMS—APPEARANCES—TREATMENT—OTHER ALKALOIDS IN OPIUM.

POISONING BY POPPIES.

THE heads of the white poppy, grown in this country, contain meconate of morphia. They yield an inspissated extract called English opium, which, according to the late Mr. Hennell, contains five per cent. of morphia. The white poppy-heads, therefore, yield to water, in the form of decoction, a poisonous salt capable of acting deleteriously on infants or children. Among several cases illustrating the noxious effects of poppy-heads to infants, the following may be mentioned: Two children in a state of narcotism were brought to a medical man. On inquiry it was found that, with a view of procuring sleep, the mother had boiled a poppy-head in water, and had given to each child, one or two teaspoonfuls of the decoction. In spite of treatment, one child died seven or eight hours after it was first seen. The decoction had the usual opiate smell. (British Med. Journal, Oct. 31st, 1857, p. 909.)

Many cases of poisoning have occurred from the injudicious use of *syrup of poppies*, which is nothing more than a sweetened decoction of the poppy-heads. This syrup, in its ordinary state, is said to contain *one grain* of extract (opium) *to one ounce* (Thomson). The common dose of it for an infant three or four months old, is half a drachm,—for an adult, two to four drachms. There is reason to believe the compound sold by some druggists for syrup of poppies, as a soothing or cordial medicine for children, is nothing more than a mixture of tincture or infusion of opium with simple syrup; it is therefore of variable strength. This may account for what appears to many persons inexplicable, namely, that an infant may be destroyed by a medicinal dose. (Med. Gaz. May, 1831, p. 253.) In January, 1841, a child six months old is said to have died from the effects of less than half a teaspoonful of syrup of poppies bought at a retail druggist's. The narcotic symptoms were fully developed in three-quarters of an hour. The syrup in this case may have contained tincture of opium. Seven children are reported to have lost their lives by this syrup in 1837–8. In one of these cases, a teaspoonful and a half was given. Stupor came on in half an hour, and the child died the following day. The late Dr. Pereira states that he has known a teaspoonful to prove fatal to a healthy child. (Mat. Med. vol. ii, pt. ii, p. 644.)

Dr. Burke Ryan communicated to me the particulars of a case which proved fatal from a similar dose in December, 1854. A drachm of the syrup was given by a mother to her child, aged fifteen months. In four hours the child was found in a deep sleep, with stertorous breathing,—the face cold and pale,—the body motionless, and in this state it died, eight hours after taking the syrup. I found, by examination, that the syrup was such as is usually sold in druggists' shops. The principal appearances in the body were congestion of the membranes and sinuses, but not of the substance of the brain. There was a reddish-colored fluid in the ventricles. The heart was firm, and the right cavities were filled with half-coagulated black blood. The stomach and intestines were healthy. (Lancet, January 20th, 1855, p. 68.)

The following are cases of poisoning by the *decoction of poppies:* A woman boiled two poppy-heads in a quarter of a pint of milk, and gave only two small spoonfuls of this decoction to her child. In an hour, the child fell into a lethargic sleep,—the respiration was stertorous, and in ten hours it died. On inspection, the brain and its membranes were found congested. In a second case, the maid-servant, in order to quiet a child, gave to it two teaspoonfuls of a decoction made by boiling one poppy-head in a small quantity of water. The child was found dead in the morning. The brain and its membranes were much congested; and the ventricles contained bloody serum. The seeds of the poppy were found in the stomach.

In the following case, the symptoms were of a serious character, but the child recovered. A woman gave to her child several teaspoonfuls of a strong decoction of poppy-heads. In a quarter of an hour it fell into a deep sleep, from which it could not be roused. Medical assistance was not called for thirty-six hours, and then the child was apparently dying. The eyes were sunk, the lids half-open and surrounded by a livid circle, the pupil *dilated* and insensible, the face pale, with a slight bluish tint, the extremities almost paralyzed, respiration hurried, the pulse frequent, small, and trembling, the forehead covered with a cold sweat, and the lower jaw depending. No urine had been voided, and there had been no evacuation from the bowels since the first occurrence of the symptoms. It was then too late to think of removing the poison from the stomach. Coffee and other stimulants were used, under which the child recovered. (Ann. d'Hyg. 1845, i, 212; also Med. Gaz. xxxvi, p. 305.)

It may be observed that the poisonous salt of morphia is generally considered to exist in the *capsule* of the poppy, and not in the seeds; but Sobernheim mentions one or two cases of poisoning by the seeds of the plant. (Tox. 500.) For other cases, see Henke Zeitschrift der S. A. 1844, i. 302. *Extract of poppies* acts like the decoction, but it is more powerful. The dose of this medicinal preparation for adults, is from two to twenty grains.

GODFREY'S CORDIAL.

This is chiefly a mixture of infusion of sassafras, treacle, and tincture of opium. The quantity of tincture of opium, according to the late Dr. Paris, is about one drachm to six ounces of the mixture, or *half a grain of opium to one ounce*, but it is very probable that, like the so-called syrup of poppies, its strength is subject to great variation. A case is reported, in which half a teaspoonful, = 1-32d part of a grain of opium, was alleged to have caused the death of an infant. In 1837–8, twelve children were killed by this mixture alone. The explanation of this is, that the opiate medicine is given in large doses by very ignorant persons.

DALBY'S CARMINATIVE.

This is a compound of several essential oils and aromatic tinctures in peppermint water, with carbonate of magnesia and tincture of opium. According to the late Dr. Paris, there are *five drops* of the tincture, or from one-quarter to half a grain of opium, in rather more than *two ounces* of this mixture, or *the one-eighth to a quarter of a grain* in an ounce. The formula commonly given is, carbonate of magnesia two scruples, oil of peppermint one minim, of nutmegs two minims, of aniseed three minims, tincture of opium five minims, spirit of pennyroyal and tincture of assafœtida of each fifteen minims, tincture of castor and compound tincture of cardamoms of each thirty minims, and of peppermint water two ounces. According to this formula, tincture of opium forms the 1-211th part by measure, or on the pharmaceutical strength, one teaspoonful would contain the 1-64th part of a grain of opium. Like most of these quack-preparations, it no doubt varies in strength. An infant is reported to have been destroyed by *forty drops* of this nostrum,—a quantity, according to the strength assigned, equivalent to more than *two minims* of the tincture, or from one-sixth to one-tenth part of a grain of opium. Accidents frequently occur from its use, partly owing to ignorance, and partly to gross carelessness on the part of mothers and nurses.

PAREGORIC ELIXIR—COMPOUND TINCTURE OF CAMPHOR.

This is a medicinal compound of alcohol, opium, benzoic acid, oil of aniseed, and camphor. Opium is the active ingredient, and of this the tincture contains rather less than *one grain* in every *half ounce* (nine grains to five ounces). It is sold to the public at the rate of fourpence per ounce. Fatal cases of poisoning by paregoric are not very frequent: the following occurred in March, 1843. A child between five and six years old had had some cough medicine prescribed for it at a druggist's. The medicine consisted, as nearly as could be ascertained from a portion left in the bottle, of paregoric, having about from one-fourth to one-half the strength of the pharmacopœial tincture. The child took two-thirds of this mixture, given in divided doses, at somewhat irregular intervals, and died in about thirty-six hours. The quantity of opium in the portion of the mixture taken was, judging by comparison with the usual strength of the tincture, from t[illegible]ourths of a grain to one grain and a quarter. The child was drows[illegible]er each dose, and slept on one occasion for several hours together. [illegible] was occasionally roused, and appeared sensible, but again relapsed into drowsiness on taking the medicine. A few hours before its death, it was found comatose, with stertorous breathing and strongly contracted pupils. On *inspection* the whole of the organs were healthy, with the exception of the parietes of the ventricles of the heart, which were somewhat thickened;—there was no congestion of the vessels of the brain, nor effusion in the ventricles. The liquid contents of the stomach yielded no trace of opium or an opiate. There was, however, no doubt that the death of this child had been caused by an opiate. This was proved, 1, by the nature of the medicine taken; 2, the nature of the symptoms, which were aggravated after each dose; 3, the confirmed coma and stertor; and, lastly, the absence of every other cause to account for the rapid death under the circumstances. (G. H. Reports, April, 1844.) It was a question here, how far a small quantity of opium in divided doses was likely to prove fatal to a child of this age? The answer was given to the effect, that although each dose might be individually harmless, the frequent repetition of the medicine, when the child had scarcely recovered from the effects of the former dose, might operate fatally. (See paper by Dr. Beck, Med. Gaz. vol. xxxiii, p. 771.) In another case a child aged 7 months was killed by a teaspoonful given in two doses at an interval of a day, *i. e.* by a dose equal to one quarter of a grain of opium. (Pharmaceut. Jour. Ap. 1845, p. 464.) Dr. Beck has recorded an instance in which a child was narcotized by about twenty drops of this tincture, = 1-12th grain of opium, or at a maximum, 1-120th grain of morphia (Medical Gazette, March, 1844, vol. xxxiii, p. 767). Dr. Edwards communicated to me in 1857 a case in which a dose of this tincture corresponding to 1-90th of a grain of opium, destroyed an infant. Aged persons may also die from the effects of small doses. At a recent inquest the deceased, a woman æt. 77, laboring under chronic cough, was proved to have died from the effects of a dose of two drachms of this liquid—equal to about half a grain of opium.

DOVER'S POWDER (COMPOUND POWDER OF IPECACUANHA).

This is a preparation of opium, the effects of which on children have been already adverted to (page 532). The proportion of opium is one-tenth part, or *one grain* in every *ten grains* of the powder. A child has been killed by four grains—therefore by a quantity containing about two-fifths of a grain of opium.

The following case of poisoning by Dover's powder is reported by Mr. Griffiths. (Medical Gazette, March, 1844.) About ten grains of the powder (equivalent to *one grain* of opium) were given by mistake to an infant 7 weeks old, and it died in twenty-four hours. On an *inspection* of the body, the countenance was placid, and the fingers of both hands were firmly contracted. In the abdomen, the spleen, kidneys, and intestines were found in a healthy condition; the liver was gorged with blood; the stomach contained a small quantity of a colorless viscid matter. The inner coat was reddened; and at the great curvature, as well as in other parts, the bloodvessels were highly injected in patches. The lungs were gorged with blood; the upper lobes being infiltrated with a greenish serum. The pericardium was reddened, and contained about a drachm of fluid. The right auricle was empty; the left ventricle contained some thin fluid blood, and a small coagulum. The sinuses of the dura mater were filled with dark coagula; the surface of the brain appeared covered by a complete network of vessels, distended with light-colored blood. On the surface of each posterior lobe there was a slight effusion of blood. The brain was soft, and the difference of color between the gray and white matter barely discernible. The vessels in the substance of the brain were gorged with blood, presenting on section, a thickly studded appearance—the spots were of a deep dull red, and in many places coalescing. There was a small quantity of fluid in each lateral ventricle, and on the floor of each the bloodvessels were largely distended. There was an effusion of serum on the surface as well as at the base of the brain, to the amount of half an ounce. The contents of the stomach were carefully analyzed, but neither morphia nor meconic acid could be detected. Although the dose here taken might be expected to prove fatal to infants or children, there is at least one instance reported in which an infant of six months recovered after having taken ten grains of this powder (Dr. Guy, in Lancet, June 8, 1850).

BLACK DROP.

This is a preparation of opium, in which the meconate of morphia is combined with acetic acid. In the Black drop, according to Pereira, verjuice, the juice of the wild crab, is employed as a menstruum instead of vinegar. The Black drop is considered to have from three to four times, and according to Dr. Neligan twice, the strength of the tincture of opium. A formula for this preparation will be found in Dr. Neligan's work, On Medicines, &c. p. 275. According to this, it is a compound of half a pound of opium to three pints of the expressed juice of the wild crab, with nutmegs, saffron, and sugar. It resembles the *Acetum Opii.* Its strength is liable to great variation.

SEDATIVE SOLUTION (BATTLEY'S).

This, according to Pereira, is an aqueous solution of opium with a little spirit and less meconic acid than the common tincture. It is considered to have three times the strength of tincture of opium, but there is so great a difference of opinion on this point, that Dr. Neligan represents it as being of only about the same strength as laudanum. (Medicines, &c. 276.) He states that it is composed of three ounces of extract of opium, six drachms of spirit, and as much distilled water as will make up two pints. It may be regarded as an aqueous solution of meconate of morphia (without the resin) and with just sufficient spirit to preserve it. It appears to be an uncertain preparation. Mr. Streeter stated at the Westminster Med. Soc. Dec. 1838, that he had known one drachm and a half of it to prove fatal to a lunatic; and twenty minims of the solution destroyed the life of an old woman. A medical gentleman, lying dangerously ill from an attack of dysentery, took, by mistake, about seven drachms of Battley's Solution. Within five minutes, salt and water, with mustard, were administered, and twenty-four grains of sulphate of zinc. Vomiting ensued; the emetic was repeated, and with the same effect; the fluid evacuated at the second vomiting having the usual smell of opium. Half a drachm of ipecacuanha was afterwards given to complete the emptying of the stomach. Notwithstanding this repeated vomiting, symptoms of narcotism presented themselves speedily, with contraction of the pupils, and very great drowsiness—rendering it necessary to remove the patient from bed in his very debilitated state, and to keep him constantly moving, until about 9 P.M. (seventeen hours), when vomiting came on spontaneously;—he was then put to bed, and allowed to sleep. The original disease afterwards resumed its course (complicated by an attack of gastritis), and at length terminated favorably; but the patient had no recollection of what had occurred for twenty-four hours after the administration of the emetics; and it appeared to his medical attendants that an excited state of the mind remained for some days afterwards. (Prov. Journ. Jan. 28, 1846, 42.) The death of Dr. Baddeley, of Chelmsford, from a medicinal dose of this solution, furnishes an additional proof of the dangerous uncertainty of the strength of this compound (see the case of Major Forester, p. 526).

WINE OF OPIUM (VINUM OPII).

This is a Pharmacopœial compound of opium, common cloves, and sherry wine. It was formerly known as Sydenham's laudanum, or *Laudanum liquidum Sydenhami.* For internal use, the dose is from ten minims to one drachm. It is considered to have about the same strength as laudanum, and its effects in poisonous doses are similar to those produced by that compound. It is rarely used as a poison. (See p. 528.)

OPIUM LOZENGES.

Some of the cough lozenges sold to the public contain a quantity of opium. Mr. Garlick communicated to the Lancet a case in which he was called to a man who had occupied himself during an afternoon in sucking one ounce and a half of these lozenges. After a time he was observed to become drowsy. His countenance was pale: there was great somnolency; and it was with difficulty that any reply to a question could be obtained. The pupils were strongly contracted; the breathing heavy and oppressed, occasionally stertorous; and the pulse small and feeble. With some difficulty, and after active treatment, the man recovered; but for a period of twenty-four hours he experienced general numbness. The vender of the lozenges knew nothing about the quantity of opium contained in them! (Lancet, 137, Jan. 30, 1847.)

MORPHIA AND ITS SALTS.

Morphia and its saline combinations must be regarded as active poisons. The pure alkaloid is known from its salts by its great insolubility in water, and owing to this property some have regarded it as less poisonous. The acid secretions of the stomach, however, dissolve it in sufficient quantity to produce speedily dangerous effects. The principal salts of morphia are the hydrochlorate or muriate, sulphate, and acetate. The first of these is now largely employed in medicine; it is preferred to the pure alkaloid, which is seldom given medicinally. These salts are used in powder and solution, but most commonly in the latter form. The medicinal dose of morphia, or of either of its salts, may be stated to be from one-eighth of a grain gradually increased to two grains. There is probably no poison more completely under the influence of habit than morphia,—hence large doses may be borne after a short time with impunity. There is a case recorded in which a physician had accustomed himself to take from fifteen to eighteen grains of the sulphate of morphia in twenty-four hours! (Dr. Myers, Ed. Med. Jour. 1855–6, vol. i, p. 357. See post, p. 547.) When applied to the skin, the cuticle having been previously removed by a blister, a dose of from one to two grains is employed: but violent symptoms have been occasionally observed to follow even from *half a grain* or less (p. 526), under these circumstances. The crystallized sulphate of morphia contains 76 per cent., and the crystallized muriate or hydrochlorate contains 88 per cent. of morphia. The sulphate of morphia is not much used in this country. The solution of the acetate is less used medicinally than that of the hydrochlorate. The dose of the former is from six to fifteen minims, and of the latter from ten minims to half a drachm. About one hundred and six minims of the solution contain one grain of hydrochlorate of morphia.

SYMPTOMS.—Dr. Vassal describes the symptoms produced by acetate of morphia used *medicinally* in the following order: 1, an irresistible tendency to sleep; 2, a well-marked slackening of the circulation, the pulse becoming small and slow, but remaining regular; 3, the contractility of the pupils, as well as the sensibility of the retina augmented; 4, copious perspiration during sleep—this ceases if the use of the salt be continued, but reappears on every increase of dose; 5, obstinate constipation; 6, more or less retention of urine; 7, irritation over the whole of the skin, accompanied in some instances by an anomalous eruption. (Considérations Médico-Chimiques sur l'Acétate de Morphine, 102.) When any one of the salts of morphia is taken at once in an overdose, the symptoms are strongly marked, and they follow each other more speedily. They generally *commence* in from *five to twenty minutes* after the poison has been swallowed; and they closely resemble those observed in acute poisoning by opium. As a summary, it may be stated, that they consist in dimness of sight, weakness and general relaxation of the muscular system, tendency to sleep, stupor, loss of consciousness, coma, stertorous breathing, and more commonly than in poisoning by opium, there are convulsions. According to Orfila, in nineteen-twentieths of all cases, the pupils will be found strongly contracted, a statement which I believe to be correct: the few exceptional cases were those in which the dose was excessive, and the symptoms were observed in the stage preceding death. The state of the pulse varies greatly. There is generally some great irritation, with itching of the skin, and irritability of the bladder, with difficulty of passing urine. Vomiting and even purging have been met with in instances in which the dose was large.

A woman gave to her child, aged 17 months, a teaspoonful of a solution of hydrochlorate of morphia by mistake for lime water. The dose was equivalent to 9-16ths of a grain of the solid salt. In half an hour the child was drowsy,—there was hurried respiration, the features were placid and composed, the skin was cold and pale, the pupils were strongly contracted. Four hours and a half had elapsed without vomiting. Coffee was given and galvanism used to keep the child roused. In about eight hours the pupils began to dilate and the child recovered. (Edinburgh Medical Journal, Feb. 1858, p. 719, case by Mr. Murray.)

Dr. Melion affirms that the acetate of morphia operates as a more powerful narcotic on children than opium. Even from small doses administered medicinally, he observed the following symptoms: the child became dull and drowsy, and gradually fell into a state of stupor; it lay with its eyes shut, or half open—one more so than the other; the ball of the eye fixed or rolling about; the pupil contracted and inactive; the heat of the head increased, and the scalp and face covered with a copious perspiration. The child murmured or spoke during its sleep, and moved its upper lip and jaw as if in the act of sucking; if it awoke, it expressed a desire to drink, and again fell asleep. It continued in this state for eight or twelve hours. In a more advanced stage, there was venous congestion over the whole body, the child lay listless, the skin was purple, the temperature diminished, the pupils strongly contracted and quite inactive, the pulsations of the heart were weak, the respiration was slow, the pulse quick or slow, small and weak, and all the secretions were suppressed. Unless quickly roused, convulsions ensued, and these were followed by death. (Monthly Journal of Medical Science, Dec. 1846, 455.)

APPEARANCES AFTER DEATH.—The only appearances which can be referred to the action of morphia, or its salts, are fulness of the vessels, with occasionally serous effusion in the ventricles of the brain and congestion of the lungs. These poisons have no local irritant action, and they, therefore, leave no marks of their operation in the stomach and bowels.

A girl, æt. 19, took in divided doses over a period of six hours, two drachms of a solution of muriate of morphia, equivalent to rather more than *one grain* of the salt. She then fell into a state of insensibility, which had continued up to the time of her admission to the hospital; the breathing was somewhat hurried and oppressed; the pulse almost imperceptible, and so rapid that it could not be counted; the pupils were contracted to the size of a pin's head. She died soon afterwards: *i. e.*, rather more than thirteen hours from the period at which she commenced taking the mixture. On inspection, the only morbid change observed in the head, was a somewhat less marked appearance of the depressions and the convolutions of the brain; and the vessels contained dark-colored blood. There were marks of inflammatory disease in the lungs. The stomach, which was much corrugated, contained a pint of grayish-colored fluid. It was perfectly healthy. In the heart and great vessels there were numerous coagula of blood. There was purple lividity of the face, neck, and arms. (Dr. Paterson, in Monthly Jour. Med. Science, Sept. 1846, p. 191.) Dr. Paterson suggests, what is not improbable, that the inflamed condition of the lungs in this case may have added to the intensity of the effects produced by the morphia. An account of the appearances produced by an overdose of the sulphate of morphia has been published by Orfila in a report of the case of Dr. Ellenberger. (Ann. d'Hyg. 1852, ii, p. 359.) The case presents some curious features. The deceased imagined that he had discovered a certain antidote for morphia and its salts, and proposed, while Orfila was at Prague, in October, 1851, to swallow the poison and the antidote in his presence. Orfila consented to witness the experiment. A powder was produced, which was found to have a bitter taste, and to possess some of the chemical properties of morphia, evidently mixed, however, with some other substance. The Doctor swallowed about twenty-three grains of this powder, and immediately afterwards, his so-called antidote, which was a fine white powder having a sweetish taste. He did not suffer from any symptoms of poisoning. Orfila, with a keen eye to the practical use of antidotes, inquired whether he had ever allowed a certain interval to pass before taking the remedy. Dr. Ellenberger said that the results were the same. About six months afterwards, Dr. Ellenberger died from a dose of about ten grains of sulphate of morphia. He had taken his antidote, but not until a considerable interval had elapsed. A minute inspection of the body was made, and the principal appearance was a well-marked congestion of the brain and its membranes. Traces of sulphate of morphia were found in the stomach. The so-called antidote was examined, and found to consist of a mixture of magnesia and carbonate of magnesia!

FATAL DOSE.—Although the salts of morphia are energetic poisons, there are several cases on record which show that they may be sometimes taken even in large doses with impunity. The conditions of the body which influence the effects of opium operate equally with respect to morphia. The *hydrochlorate* has been given in doses of *two grains* every six hours without ill effects; but then the commencing dose was a quarter of a grain, and this was gradually increased. Under these circumstances, it is well known that, after a time, as much as from eight to ten grains will be required to act as a narcotic. In diseased conditions of the nervous system, doses of these salts which are usually regarded as poisonous, may be given without injury. Dr. Pereira states that, in a case of insanity, he gave *two grains* of the muriate at one dose, without any ill effects following. The following illustration of a tolerance of the salts of morphia is still more remarkable. It occurred in the practice of Mr. Kellock in June, 1857, and was communicated to me by Dr. Rees. Thirty grains of the hydrochlorate of morphia were given in twenty-four hours to a lady laboring under puerperal mania, without any symptoms of poisoning being manifested! I examined the salt of morphia and found it to be of the usual pharmaceutical purity.

In one instance an adult, who had taken the hydrochlorate medicinally, was killed by a dose of three grains. In a case which occurred in London, in April, 1846, a woman, æt. 66, was killed by a dose of six grains, under the following circumstances: She was admitted into University College Hospital, laboring under gangrene of the right foot. A draught containing three quarters of a grain of muriate of morphia was prescribed for her: she had taken this dose, it appears, safely on two or three previous occasions. By some mistake, a stronger solution of morphia than that intended by the prescriber was used, and a draught, containing *six grains* of the muriate, was given to her by the nurse about nine o'clock. In three hours, the woman was found in a state of complete narcotism, and in spite of every available remedy, including galvanism, she gradually sank, and died *nine hours* after taking the draught. (Med. Times, May 9, 1846, p. 114.) Dr. Christison has collected three cases, in each of which ten grains of the muriate proved fatal. In one of these, a woman took this dose of the pure muriate by mistake. This was discovered immediately, and means were used to prevent any ill effects from the accident. Within half an hour after the poison had been swallowed, the stomach was completely cleared by the stomach-pump. At this time the patient was quite sensible. Stupor quickly came on,—deep coma gradually appeared; and before night she expired. (Op. cit. 727.) This case is important, because it shows,—first, that *absorption*, to an extent to prove fatal, may occur in *half an hour;* secondly, that there may be a complete remission in the symptoms and afterwards a fatal relapse (see page 523); and, thirdly, as no poison was found in the body, it throws an important light upon the evidence from chemical analysis, as it proves that a person may die after many hours from the effects of a large dose of morphia, and no trace of the poison remain in the stomach.

The *acetate of morphia* is subject to similar anomalies with respect to dose. Mr. Headland states that he attended an opium-eater, who was in the habit of taking seventeen or eighteen grains of the acetate daily. There is a case on record, in which a young man swallowed ten grains of the acetate, and shortly afterwards forty grains: he suffered from the usual symptoms, but ultimately recovered, although he had taken in the whole fifty grains of the acetate of morphia on an empty stomach! An account of this case will be found in Schneider's Ann. der S. A. 1836, i, 455. In the Edinburgh Medical and Surgical Journal (vol. xxxiii, p. 220), a case is reported in which a young man recovered in four days, after taking *twenty grains* of the acetate; but the most remarkable instance of all is that reported by M. Bonjean, in which a young man entirely recovered in about eight hours from a dose of *fifty-five grains* of acetate of morphia in an ounce of water. No symptoms of any importance manifested themselves, until *an hour* after the poison was taken, and then there was simply giddiness with a tendency to sleep. Two hours after the occurence, he still had the power to answer questions! In four hours deep coma supervened, but under copious bleeding and other treatment, this gradually disappeared. (Ann. d'Hyg. 1845, i, 150.) A man swallowed at once *ten grains and three-quarters* of acetate of morphia. Tartar-emetic was immediately given, but without producing vomiting. About three hours after the accident, and while the patient was in a state of deep coma, a highly concentrated solution of coffee with the solid residue was given to him. The coma disappeared, and he perfectly recovered. (Gaz. Méd. Mai, p. 346.)

On the whole, notwithstanding these recoveries from large doses, we are justified in regarding it as rather the exception to the rule, that a person not used to the drug, should escape, who has taken more than *two grains* of either salt, and that a smaller quantity than this will suffice to kill a child, or in some cases an adult. Dr. Vassal states that, when he gave in one day more than *three grains* of the acetate to a patient, narcotism was invariably induced. Symptoms of poisoning have certainly been produced by *one grain* of the acetate; and that the hydrochlorate even, when *one grain* is given in divided doses, may destroy life, is proved by the case reported by Dr. Paterson (page 545). This is the smallest fatal dose of the salts of morphia yet known, and the result is corroborated by the fact that Dr. Kelso, in experimenting upon himself, found, that half a grain of the hydrochlorate produced very severe symptoms. (Lancet, Sept. 1839.) The sulphate and the acetate would, no doubt, act with equal power. In the Lancet for November, 1838, a case is related in which only *half a grain* of the acetate was supposed to have caused the death of a lady, to whom it had been administered as medicine. She was at the time in ill health.

The poisonous action of the salts of morphia gave rise to an important medico-legal investigation in Paris, in 1823, which ended in the trial and execution of a Dr. Castaing, who had formerly been a pupil under Orfila. He was charged with having made use of his knowledge of poisons to take away life. The facts were these: Dr. Castaing was intimate with two brothers, who were young men of fortune. One of them, Hippolyte Ballet, who had been for some time an invalid, and was attended by Castaing, died in October, 1822. Although he died rather suddenly, and under severe symptoms, no suspicion arose that his death had occurred from other than natural causes. On inspection, nothing was observed in the body to indicate a violent death. He made his will in favor of Castaing, and made no bequeathal to his brother. Castaing afterwards surrendered the will to the surviving brother for a large sum of money. About seven months afterwards the prisoner Castaing, who had been travelling about with the surviving brother, Augustus Ballet, unaccompanied by servants, put up at a tavern at St.

Cloud. In the evening Ballet complained of feeling unwell: some sugared wine was given to him by Castaing, but without relief. The prisoner left his companion at four o'clock in the morning, as he said to take a walk in the park; but it was proved that he privately returned to Paris, procured at the shop of one druggist twelve grains of tartar emetic, and at another shop *twenty-six grains* of acetate of morphia. He immediately returned to Ballet, whom he found still lying ill. He prescribed for his patient cold milk, and gave it to him himself. In five minutes Ballet was seized with convulsions, and in about half an hour with vomiting and purging. He was attended by a physician, who treated the case as one of cholera morbus. In the evening the patient became insensible; he was unable to swallow; his skin was bathed in a cold perspiration; a small pulse; he had jaws locked, the neck rigid, the abdomen tense, and there were convulsions of the extremities. After a few hours he was seen by another physician: the breathing was then stertorous, and the pupils much contracted: there was an entire loss of sensibility in the legs. He died about thirty-two hours after the commencement of the symptoms in a severe form. On inspection the chief morbid appearance was congestion of the vessels of the brain, with serous effusion beneath the membranes. The contents of the stomach were analyzed by Vauquelin and Barruel, but no trace of any poison could be detected. Owing to the strong moral evidence against him, the prisoner was put on his trial for the murder of the deceased; and the most eminent medical jurists in France gave evidence on the occasion. The opinions of Orfila, Magendie, Vauquelin, and Laennec, went to show that the symptoms and death might be referred either to vegetable poison or to natural disease. The absence of poison in the body might have been due to vomiting, absorption, and the difficulty of detecting the salt of morphia. (Guy's Hosp. Reports, Oct. 1856; Smith's Analysis of Med. Ev. p. 368.) In spite of the absence of chemical evidence, the prisoner was properly convicted.

Treatment.—In poisoning by morphia or its salts, the same treatment is required as in poisoning by opium (p. 535). For the *Chemical Analysis*, see p. 550).

OTHER ALKALOIDS IN OPIUM.

Besides morphia, opium contains three alkaloids, Codeia, Narcotina, and Thebaina,—some neutral principles, Porphyroxine, Narceine, and Meconine, and one organic acid, the Meconic.

NARCOTINA.—The results of experiments with this substance on animals are very conflicting. In the human subject it has been observed to produce headache; but when these effects have followed, it has been probably mixed with morphia. I have frequently found this mixture in specimens: and Dr. Christison states, that he has met with narcotina in morphia, a circumstance, which may tend to explain the variable effects of morphia in large doses.

CODEIA is found to exert a poisonous action on animals: it has been used in France as a narcotic. It is considered to have only one-half of the strength of morphia, with which it is in general mixed. The common hydrochlorate of morphia of the shops is, according to Pereira, a compound of morphia and codeia. M. Kunckel found that, when combined with acids, it lost much of its activity.

THEBAINA (PARAMORPHINE).—According to Magendie, this substance, when injected into the jugular vein of an animal, acts like strychnia, producing tetanus and death in a few minutes. Orfila found that it produced opisthotonos, but the animals recovered. (Op. cit. ii, 203.)

The other constituents appear to be inert. Experiments hitherto made show that they exert no poisonous action on the animal system.

CHAPTER XXXV.

CHEMICAL ANALYSIS OF OPIUM—EVIDENCE FROM ODOR—MORPHIA—PROPERTIES IN THE SOLID STATE AND IN SOLUTION—MECONIC ACID—ITS PROPERTIES—TESTS FOR NARCOTINA, CODEIA, PORPHYROXINE, AND NARCEINE—DETECTION OF OPIUM IN ORGANIC MIXTURES—OPIUM RARELY DISCOVERED—OBJECTIONS TO THE CHEMICAL PROCESSES FOR MORPHIA AND MECONIC ACID IN ORGANIC LIQUIDS—MORPHIA IN THE TISSUES—QUANTITATIVE ANALYSIS—PROPORTION OF OPIUM IN OPIATE MEDICINES.

CHEMICAL ANALYSIS.

OPIUM.—There are no means of detecting opium itself, either in the solid or liquid state, except by its smell and other physical properties, or by exhibiting a portion of the suspected substance to animals, and observing the effects produced. Independently of alkaloids and principles, opium contains gum, resin, coloring matter, and other vegetable substances in variable proportion. The smell is said to be peculiar, but a similar smell is possessed by lactucarium, which contains neither meconic acid nor morphia. The *odor* is, however, a good concomitant test of the presence of the drug, whether it be in a free state or dissolved in alcohol or water, but it is not perceptible when the solution is much diluted or has been long exposed. I have found that half a grain of powdered opium, dissolved in half an ounce of water, lost its characteristic smell by a short exposure to air. The odor is decidedly volatile, and passes off when an opiate liquid is heated; it also escapes slowly at common temperatures. Again, it may be concealed by other odors, or the drug may undergo some change in the stomach during life which may destroy the odor. The analysis, in case of poisoning by opium, is therefore limited to the detection of morphia and meconic acid.

Morphia.—Morphia is known by the following properties: 1. It crystallizes in fine quadrangular prisms, which are white and perfect, according to their degree of purity. 2. When heated on platina, the crystals melt, become dark-colored, and burn like a resin with a yellow smoky flame, leaving a carbonaceous residue. If this experiment is performed in a small reduction-tube, it will be found, by employing test-paper, that ammonia is one of the products of decomposition. 3. It is scarcely soluble in cold water, requiring 1000 parts to dissolve it; it is soluble in one hundred parts of boiling water, and the hot solution has a faint alkaline reaction. By its insolubility in water it is known, and may be separated from its salts. It is not very soluble in ether, thus differing from narcotina and some other alkaloids; but it is dissolved by forty parts of cold, and rather less than this quantity of boiling alcohol. It is soluble in potash, soda, and lime water, but less in ammonia. Owing to the great solubility of this alkaloid in alkalies, it is not readily precipitated from its saline solutions. It is not dissolved by chloroform or benzole. 4. It is easily dissolved by a small quantity of all diluted acids, mineral and vegetable. 5. Morphia and all its salts have a bitter taste. In order to apply the chemical tests for morphia, a portion may be dissolved in a few drops of a diluted acid, which may be either the acetic or muriatic. If either the muriate or the acetate be presented for analysis, a portion may be at once dissolved in a small quantity of boiling water.

TESTS.—The tests for this alkaloid are the following: 1. *Nitric acid.* This, when added to a moderately strong solution of a salt of morphia, produces slowly a deep orange-red color. If added to the crystals of morphia or its salts, deutoxide of nitrogen is evolved: the morphia is entirely dissolved, and the solution acquires instantly a deep orange-red color,—becoming, however, much lighter by standing. In order that the effect should follow, the morphia must be either in the solid state, or if in solution, not too much diluted, and the acid must be added in pretty large quantity. The color is rendered lighter by boiling: therefore the test should never be added to a hot solution. 2. *Perchloride of iron* (sesquichloride), or colorless persulphate. Either of these solutions, when saturated and neutralized (by a small quantity of potash, if necessary), gives an inky-blue color to a solution of morphia, or to the solid crystals, if not too acid. If the quantity of morphia is small, or there is any free acid in the test or solution of morphia, the color is greenish-blue. The blue color is removed by acids, but restored by alkalies—it is also destroyed by heat: thus the iron-test should never be employed with a very acid or a very hot solution of salt of morphia. The blue color given by this test in a solution of morphia, is entirely destroyed by nitric acid and replaced by the orange-red color, so that the nitric acid will act through the iron test, but not *vice versâ*. In this way two tests may be applied to one quantity of liquid. 3. *Iodic acid.* Morphia or its salts in the solid state or in solution decompose this acid, taking parts of its oxygen, and setting free iodine. The liquid becomes brown and smells of iodine, and on adding to it a solution of starch, it acquires a blue color. If the quantity of morphia is small, there is only a reddish or purple tint slowly produced: if large, the dark-blue iodide of farina is formed in a few seconds. As this color is destroyed by heat, the test must not be added to a hot solution. 4. *Sulphuric acid* and *bichromate of potash.* When strong sulphuric acid is poured on pure morphia in a solid state, there is either no effect, or the alkaloid acquires a light pinkish color. On adding to this a crystal of bichromate of potash, the mixture speedily becomes green (from oxide of chromium), and retains this color for some time. The solutions of morphia are, like those of other alkaloids, precipitated by tannic acid, but not by gallic acid.

Of the tests here selected, it may be remarked that the nitric and iodic acids act with great delicacy upon almost any visible quantity of morphia or its salts in the solid state. Dilution with water materially affects the reactions on a solution of morphia; hence it is desirable to operate either on the solid crystalline residue, however small, or on a highly concentrated solution. Morphia may be obtained perfectly crystallized for microscopic observation, by dissolving a small portion on a glass slide in diluted hydrochloric acid, and then adding a drop of a weak solution of ammonia. Brucia, strychnia, veratria, and other alkaloids thus treated, produce very different forms. Brucia and common strychnia give, like morphia, a deep red color with strong nitric acid, but they are unchanged by iodic acid; and strychnia is differently affected by sulphuric acid and bichromate of potash. When we are dealing with the solid and pure alkaloid, it may be easily distinguished from all others. The acids of the salts of morphia may be identified by their respective tests.

Meconic acid.—This is a solid crystalline acid, seen commonly in scaly crystals of a reddish color. It is combined with morphia in opium, of which it is considered to form on an average six per cent.; and it serves to render that alkaloid soluble in water and other menstrua. It is dissolved by one hundred and twenty-five parts of cold water: it is much more soluble in boiling water, but is in great part precipitated on cooling. The cold saturated solution has, notwithstanding its sparing solubility, a strongly acid reaction. The solution, when very much diluted, is precipitated of a yellowish-white color by acetate of lead (meconate of lead); and this precipitate is insoluble in acetic acid—a property which allows it to be thus easily separated in analysis, not only from some of the organic compounds of the oxide of lead, but also from the sulphocyanate of lead, which is quite soluble in acetic acid.

Many tests have been proposed for meconic acid; there is only one upon which any reliance can be placed, namely, the *Perchloride* or *Persulphate of iron.* This test gives, even in a diluted solution of meconic acid, a deep red color; and it is owing to the presence of this acid, that a salt of iron causes a red color in the tincture or infusion of opium, as well as in all liquids containing traces of meconate of morphia,—the effect of the iron test on morphia being counteracted by the presence of meconic acid. The 1-500th part of a grain of this acid dissolved in a small quantity of liquid, may be detected by the iron test. The red color of the meconate of iron is not easily destroyed by diluted mineral acids, by a solution of corrosive sublimate, or by chloride of gold, but it is, by sulphurous acid and chloride of tin. When the crystals of this acid are heated, or its aqueous solution is boiled, it is partially decomposed,—a new acid is formed (comenic), and by a still higher degree of heat, pyromeconic acid is produced. The three acids, however, give the same reaction with a persalt of iron. Nitrate of silver added to a solution of meconic acid, precipitates a yellow-white meconate of silver which is dissolved by nitric acid, and when this acid solution is boiled, cyanide of silver is produced. On evaporating to dryness and heating the residue, it is not easy to procure the usual evidence of cyanogen by combustion, hence this reagent is of little practical value as a test.

Various objections have been made to a reliance on the color produced by a persalt of iron, as evidence of the presence of meconic acid. Thus *sulphocyanic acid* or an alkaline *sulphocyanide* produces a similar color with a persalt of iron, but the red color in this case, is immediately destroyed by a few drops of a solution of corrosive sublimate, or of chloride of gold. The liquid may be diluted, and a weak solution of acetate of lead added;—a precipitate falls, either as meconate or sulphocyanate of lead. The former is insoluble, while the latter is quite soluble in acetic acid. Strong *acetic acid*, or any of the alkaline acetates in a concentrated solution, also give a red color with a persalt of iron, and this color is not removed by the metallic chlorides just mentioned, in which respect these salts resemble a solution of meconic acid; but if previously boiled with a few drops of diluted sulphuric acid, the acetate gives no color, and is thus known from a meconate.

Narcotina.—This alkaloid is not commonly sought for in medico-legal investigations, but it is proper to state the chemical differences which exist between it and morphia. Its crystals are rhombic prisms, and have a bright pearly lustre. It is not very soluble in water, but it is dissolved by boiling alcohol and ether. Its solutions have no alkaline reaction, but are very bitter. Unlike morphia, it is not very soluble in potash, ammonia, soda, or lime-water, all of which precipitate it from its solutions,—or in diluted acetic acid even on boiling. When nitric acid is poured on the crystals, they acquire a *yellow*, not an orange-red color, like morphia. Sulphuric acid gives to narcotina a bright sulphur-yellow color: to morphia a pinkish-brown tint. If to the mixture of acid and alkaloid, a crystal of bichromate of potash be added, green oxide of chromium is set free in both cases, but very slowly in the case of narcotina. If to the mixture of sulphuric acid and the alkaloid, a grain of nitre is added, a deep blood-red color is slowly brought out with narcotina, but not with morphia. Narcotina does not decompose iodic acid, or set iodine free. If narcotina be boiled with acetic acid, and a strong solution of hypochlorite of lime be added, the liquid acquires at first a yellow and then a red color: morphia under the same circumstances acquires a yellow color. This mode of testing, proposed by Flandin, is not very satisfactory, as the results depend much on the proportions added. When heated on platina, narcotina, like morphia, melts and burns, and if not overheated, sets into a crystalline mass on cooling.

CODEIA.—This alkaloid, which is not often seen so well crystallized as morphia and narcotina, is known from both by its ready solubility in water, and by its forming a strongly alkaline solution. One hundred parts of water at 60° dissolve one part and a quarter; at 212° nearly six parts. It is soluble in alcohol, and combines with acids. It differs from morphia in not decomposing iodic acid, and in not giving any red color with nitric acid, either as a solid or when dissolved in acids. It differs from narcotina in not being turned yellow, but of a light pinkish-brown color, by sulphuric acid; but it resembles both morphia and narcotina in producing green oxide of chromium when a crystal of bichromate of potash is added to the mixture. When codeia is boiled with acetic acid and hypochlorite of lime, it produces a yellow color like morphia. Heated on platina it melts, forming a globule of colorless liquid; this soon darkens, and gives off a vapor which burns with a yellow smoky flame.

PORPHYROXINE.—Porphyroxine is a principle which has been described by Merck. It is soluble in alcohol, ether, and diluted acids. Its solution in a mineral acid, is reddened when heated, and alkalies precipitate from the colored solution, colorless porphyroxine. (Böcker, Vergiftungen, 1857, p. 102.)

NARCEINE.—Narceine is met with in fine prismatic crystals. It is very insoluble in water, hot or cold: the hot solution has a faint alkaline reaction. It is easily dissolved by acetic acid and by caustic potash. Strong sulphuric acid turns the crystals of a blackish-green color. If the acid be diluted with a small quantity of water the liquid acquires slowly a light blue color. On boiling it with more water it becomes colorless, but, by evaporation, it acquires a pinkish-red hue. Strong nitric acid dissolves the crystals, forming a light yellow solution.

Detection of opium in organic mixtures.—Opium may be regarded as an organic solid, containing the poisonous salt, the constituents of which we wish to extract. It is not often that, in fatal cases of poisoning by opium or its tincture, even when taken in large quantity and death is speedy, that we can succeed in detecting meconate of morphia in the stomach. It is probably removed by vomiting or absorption,—but not always by decomposition; for I have found meconate of morphia in strong opiate mixtures in water, after they have been kept for twelve or fourteen months and allowed to decompose spontaneously under the free access of air.

Non-detection of opium or its poisonous salt.—In the case of a young woman who died five hours after taking two ounces of laudanum, Dr. Christison did not succeed in detecting morphia by any of the tests. Other cases of a similar kind are mentioned by him. (On Poisons, p. 697.) In several instances of poisoning which have occurred to myself by opium, there has not been a trace of meconic acid or of morphia in the contents of the stomach. In one case, a woman swallowed an ounce and a half of laudanum in beer. In half an hour she was in a state of profound coma, and she died in nine hours. None of the poison could be detected in the stomach,—there was not even the smell of opium. In two cases, which occurred in 1844, one having proved fatal in five, and the other in twenty-two hours, there was not the least trace of opium either by the odor or by tests. In the latter case half an ounce of the tincture had been taken. (See pp. 527, 528, 541, 546, 548, and Casper, Ger. Med. i, p. 455.) The cause of the failure of chemical evidence on these occasions is partly due to the smallness of the quantity which may remain in the body at the time of death, or to its entire removal, by absorption, digestion, or decomposition, from the stomach (p. 178). Infants have been killed by doses of opium equivalent to less than the hundredth part of a grain of morphia (p. 181). To suppose that any portion of this could be separated after death from the body of a child that had survived eighteen hours would be absurd; yet some chemists have been found who could shamelessly deceive the public by swearing that no person could die of poison, except the poison remained in, and was visibly separable from the stomach or tissues after death. A statement thus publicly made and plausibly sustained by analyses undertaken for the express purpose, tends to create a difficulty in medical evidence in all cases of the poisoning of infants or adults by small doses. Dr. Ogston communicated to me a case which was tried at the Aberdeen Autumn Circuit, 1853, in which a child died in a few hours from the improper administration of a teaspoonful of the wine of opium. No trace of morphia or meconic acid could be found in the stomach, and on this fact the defence partly rested. It was properly explained that opium was liable to be removed from the stomach. The prisoner was, on this occasion, convicted. In Reg. v. Kelly (Liverpool Autumn Assizes, 1857), the prisoner was charged with administering to her child, æt. 4, two drachms of laudanum. That the child had died in a few hours under all the symptoms of poisoning by opium was made clear by the medical evidence; but Mr. Stone, who made the analysis, stated that he failed to discover morphia: he obtained results indicating the presence of meconic acid, but he declined to swear from them that opium was present. The learned judge who tried this case (Watson, B.), is reported to have said,—"If the medical witness could not swear to the *presence of opium*, how could the jury come to the conclusion that the child was poisoned by laudanum? Without the discovery of poison in the body it was always a most dangerous thing to convict!" On this the prisoner was found not guilty. (See on this subject, Poisoning by Strychnia, 1856, p. 57; also, Pereira's Materia Medica, vol. ii, pt. 2, p. 614, and Casper, Ger. Med. i, 457.) All respectable authorities agree on this question, and reject as an untrue and dangerous dogma the statement that when death has been occasioned by a vegetable alkaloid, it must invariably be found in the dead body. Dr. Pereira states that he has met with several instances in which persons have been poisoned by opium, and no trace of the drug has been discovered after death in the alimentary canal (supra). It may be set down as the exception to the rule to find this poison in the dead body. In some instances I have found traces of meconic acid, but no satisfactory evidence of the presence of morphia.

The poison may in general be detected more easily in the matter vomited during life (if vomiting should have occurred) than in the contents of the stomach after death. It was thus detected readily in the form of aqueous infusion, in a case in which the contents of the stomach had been ejected about seven hours after the poison had been swallowed. (Med. Gaz. xxxvii, p. 724.) Dr. Skae detected traces in the stomach in one instance, in which probably only two drachms of the tincture had been taken.

Various processes have been suggested for the detection of opium in organic liquids, but before resorting to any of these it is desirable to apply trial tests to the suspected liquid, in order to determine whether any meconic acid or morphia be present or not. The smell of opium may be entirely absent. The best trial tests are nitric acid and the perchloride of iron. These will produce in the infusion or liquid, if it contain opium, the changes already indicated. In testing for meconic acid, it is advisable to dilute the organic liquid, if colored, with a sufficient quantity of water to render the production of a change of color by the test perceptible. In respect to the detection of the meconate of morphia in a suspected liquid, it is proper to observe, that nitric acid will indicate the presence of morphia, and a persalt of iron the presence of meconic acid, in infusions containing so small a quantity of opium as not to be precipitated by the acetate of lead.

If the matter is solid, it should be cut in small slices; if liquid, evaporated in a water-bath to an extract; and in either case, digested with distilled water and a very small quantity of a diluted acid for one or two hours at a gentle heat. Acetic, oxalic, tartaric, and hydrochloric acids, have been employed by different analysts. For the separation of meconic acid, acetic acid is to be preferred. The aqueous acid solution should be filtered, a little acetic acid added, and then treated with acetate of lead until there is no further precipitation. The liquid should be boiled and filtered; meconate of lead is left on the filter, while any morphia passes through under the form of acetate. The surplus acetate of lead in the filtered liquid (containing the morphia) should now be precipitated by a current of sulphuretted hydrogen gas,—the sulphuret of lead separated by filtration, and the liquid evaporated at a gentle heat, in a water-bath, to an extract, so that any sulphuretted hydrogen may be entirely expelled. On treating this extract with alcohol, the acetate of *morphia*, if present in a sufficient quantity, may be dissolved out and tested. The *meconate* of lead left on the filter may be decomposed by boiling it with a small quantity of diluted sulphuric acid; and in the filtered liquid (neutralized if necessary by an alkali) the meconic acid is easily detected by the iron test. This analysis requires care as well as some practice in the operator, in order that the morphia should be obtained in a sufficiently pure state for the application of the tests, but the quantity present in the liquid may really be too small for separation by this process.

The detection of meconic acid is attended with less difficulty. It has been found in no vegetable substance excepting opium; hence, before pronouncing this drug to be present, the presence of this acid in a suspected liquid should be clearly determined. It has been erroneously asserted that laburnum, senna, dandelion, cloves, and other vegetables, contained principles which might by their reactions be mistaken for morphia or meconic acid. This, however, is not the case;—they contain no meconic acid, and no alkaloid resembling morphia.

Various modifications of the process above described have been suggested. Thus, instead of evaporating the filtrate containing the morphia, some analysts have merely concentrated the liquid and added ammonia in slight excess. A whitish or colored precipitate falls down (morphia and narcotina). If much colored, it should be dissolved in a minimum quantity of hot water, barely acidulated with hydrochloric acid, and set aside to crystallize. On cooling the liquid, prisms of hydrochlorate of morphia, sufficiently pure for testing, may sometimes be procured. If no crystals are deposited, the liquid should be still further concentrated, either by spontaneous evaporation or in a water bath. If again no crystals are formed, the dissolved alkaloids may be precipitated by adding carbonate of potash and warming the liquid. The precipitate may then be tested. It is better for the purposes of testing to procure a small quantity of morphia in a pure state, than to have a large quantity in an impure state.

Narcotina may be separated from the ammoniacal precipitate by ether; and its presence may be known by the blood-red color which is slowly produced on the addition of sulphuric acid and a crystal of nitre.

Instead of decomposing the precipitated *meconate of lead* by sulphuric acid, some analysts have found it preferable to diffuse the precipitate in water and then decompose it by a current of sulphuretted hydrogen gas, the surplus gas being subsequently drawn off by heating the liquid in a water-bath.

Merck recommends, for the detection of *porphyroxine*, that a solution of potash should be added to the concentrated solution of an opiate mixture, and the alkaline liquid then well shaken with pure ether, which dissolves and separates the porphyroxine. If a piece of filtering paper be dipped in the ethereal liquid several times, and dried between each dipping, a sufficient quantity of this principle may be fixed in the paper for the purpose of testing. The paper is simply moistened in hydrochloric acid, and held in the vapor of boiling water. If it acquires a reddish color while thus exposed, this may be taken as an indication of the presence of porphyroxine.

For the separation from organic mixtures of *morphia*, or any of its salts, *e. g.*, the acetate or sulphate, very diluted hydrochloric acid (three drops of strong acid to one ounce of water) may be used for the first aqueous extract. The residue, evaporated on a water-bath, may be then treated with hot alcohol, and the morphia, or its salt, obtained by evaporation. If the presence of hydrochlorate be suspected, then the water may be slightly acidulated with sulphuric or acetic acid. In one case of death from hydrochlorate of morphia, I found the poison in the stomach by simply employing hot alcohol. Duflos advises that the acid liquid (holding the morphia) should be neutralized by magnesia, and the morphia afterwards separated by alcohol from the precipitate thus obtained; but if hydrochloric acid is employed, the alcohol will dissolve some chloride of magnesium.

Stas's Process.—M. Stas, a Belgian chemist, has recommended the employment of ether for the extraction of *morphia* in organic mixtures containing opium. He procures, at a temperature not exceeding 160°, an alcoholic extract of the substance requiring examination, mixing with it a small quantity of oxalic or tartaric acid for the purpose of rendering the alkaloid soluble. The acid extract is filtered, and the residue exhausted with strong spirit. This is concentrated *in vacuo* by the aid of sulphuric acid, or at a temperature not exceeding 100°. The liquid is filtered through a wet filter, to separate any fatty matters dissolved by the spirit; and is again evaporated *in vacuo* nearly to dryness. The residue is now exhausted with absolute alcohol, this alcoholic liquid is evaporated, and the acid residue dissolved in a minimum of distilled water. To this acid aqueous solution,—finely powdered and pure bicarbonate of soda or potash is cautiously added, until there is no longer effervescence and the liquid is neutralized. It is then shaken in a tube with from four to six times its volume of pure rectified ether. The stratum of ether which floats on the liquid is subsequently poured off and evaporated,—when crystals of the alkaloid, if present in sufficient quantity, may be obtained. In reference to this process, I can corroborate the statement of Otto, that it is not satisfactory for the detection of morphia, in consequence of the great insolubility of this alkaloid in ether. It has been suggested by Polstorff that the ether must be added immediately after the alkaline carbonate, and the supernatant liquid poured off as quickly as possible. If the morphia is once separated in a crystalline state, as a result of the addition of the carbonate of soda, before the ether is added to the liquid, the morphia is not dissolved by the ether, or only in traces. This is, therefore, as we have found it in practice, a process of considerable uncertainty. The quantity of morphia removed by the ether is small—the larger proportion is retained by the alkaline liquid. Poellnitz has suggested that the addition of a small quantity of alcohol to the ether improves its solvent powers. Morphia is quite soluble in pure soda or potash, and ether cannot extract it from these alkaline solutions.

It will be perceived that Stas's process fails in one important point. It makes no provision for the detection of *meconic acid:* but the presence of this acid must be determined before we can infer that *opium* was present in the substance examined. If morphia alone be found, this may be ascribed to the presence of that alkaloid or of one of its salts.

Whatever process may be adopted for the extraction of morphia, either from the contents of the stomach or from an organic liquid, it is proper to bear in mind that the liquid should not be too strongly acidulated, and that it should not be heated above the temperature of a water-bath. I have found that there was no carbonization or destruction of morphia by evaporating a liquid to dryness, and keeping it in a dry state over a water-bath when the proportion of hydrochloric acid was not greater than from three to six drops to an ounce of water, while this proportion of acid formed a perfect solvent for morphia. In some recent experiments, I have substituted phosphoric acid as a solvent in the proportion of from six to ten drops of the strong acid to an ounce of distilled water. There is in this case no carbonization by concentration at a water-bath heat. In employing this acid, lime water may be used for neutralization: phosphate of lime and morphia are thrown down: the morphia may be removed by alcohol, and on evaporation, digested in weak hydrochloric acid and obtained as crystallized hydrochlorate for testing. Having precipitated a solution of phosphate of morphia by lime, I found that ether, added in an equal volume to the liquid, barely removed a trace of the alkaloid discoverable by nitric acid, while on testing the watery liquid with the precipitate, the phosphate of lime was dissolved, and the liquid, by the color produced, indicated the presence of the greater proportion of the morphia. Chloroform and benzole cannot be substituted for ether, as these exert but little solvent action on morphia.

From the small proportion of morphia and meconic acid contained in opium, it is obvious that unless the soluble matter of several grains of opium be present, it will not be easy to separate any morphia or meconic acid from an organic liquid. If the quantity of opium be less than half a grain, acetate of lead will not precipitate any perceptible quantity of meconate of lead, and it would require from one quarter to half a grain of dry meconate of lead to procure good evidence of the presence of meconic acid. When the quantity of meconate of lead is small, it should be placed in a watch-glass, covered with a few drops of diluted sulphuric acid, and gently warmed. Having allowed time for the subsidence of sulphate of lead, the persalt of iron may be added, and the change of color noticed.

In some experiments with *powdered opium*, it was found that the soluble part of one-tenth of a grain in one drachm of water, was clearly affected by nitric acid; but when mixed with one ounce of water it was not. In the last case, however, meconic acid was distinctly indicated by the action of a persalt of iron; but the tenth of a grain of opium in an ounce of water, gave with acetate of lead, no precipitate of meconate of lead, and the same quantity in one drachm of water, gave only a faint precipitate which would scarcely admit of being collected. The meconic acid may be easily determined to be present by the iron test, in a liquid containing only *one grain* of opium if not too much diluted: but it would be difficult in most cases to obtain so small a quantity of precipitate as this would yield, in a state to allow of the separation of the meconic acid. So, nitric acid might indicate morphia, when the quantity of opium in a liquid amounted to one grain or less, in which case the proportion of morphia might vary from one-fourteenth to one-twentieth part of a grain. This would not admit of easy separation: hence, unless we obtain a tolerably free precipitate of meconate of lead, insoluble in acetic acid, it will not be in our power to obtain the morphia and meconic acid separately. On the whole, it is obvious that the tests for opium and the processes for separating its poisonous salt from organic liquids are far inferior in delicacy and certainty to those employed for mineral as well as some vegetable poisons.

As there is no medicine so frequently prescribed as opium in ordinary disease, an analyst must remember that the discovery of a small quantity in the stomach is not sufficient to establish the fact of poisoning. It may be the residuary quantity of an opiate medicine lawfully prescribed for the deceased.

Objections to the processes for opium in organic liquids.—It need hardly be observed that the tests for morphia should not be applied to liquids which contain organic matter. A decoction of mustard-seed produces with two of the tests,—the nitric and iodic acids,—changes similar to those produced by morphia; while with a persalt of iron it produces a red color resembling that caused by meconic acid. An infusion or decoction of nux vomica is reddened by nitric acid, and there are many common vegetable infusions and decoctions (pimento, cloves, senna, &c.) which acquire a red or orange-red color when nitric acid is added to them. Again, the persalts of iron give a bluish color with tea, beer, and numerous organic liquids containing gallic or tannic acid. Iodic acid is decomposed by a still larger number of substances, including putrescent animal or vegetable matter, and all deoxidizing agents. Owing to an undue reliance upon these tests, as applied to organic liquids, some serious mistakes have been already made. MM. Ruspini and Cogrossi found that a decoction of the intestines of a calf, although no morphia was present, acted on iodic acid like a solution of morphia (Orfila, Toxicologie, vol. ii, p. 232.). In another case, morphia was pronounced to be present in urine by reason of the action of the extract of this liquid on iodic acid. The effect was found to be due to the presence of uric acid or urate of ammonia. In a recent case of sudden death from apoplexy, an undue reliance on the color produced by nitric acid, in an organic liquid, led to a serious error. Mr. Davey, of Romford, first called the attention of the profession to this case. A decoction of the contents of the stomach of a female was treated with nitric acid,—a red color, resembling that produced by morphia resulted, no morphia was separated, and no meconic acid was found or even looked for. Upon this questionable result, Dr. Letheby, who made the analysis, deposed at the inquest, held in July 1857, that he had found in the stomach "distinct traces of morphia,"—that the quantity which he detected was about one-tenth of a grain, and that this was equivalent to one grain of opium. Although not a fatal dose, he accounted for the deficiency by absorption, &c., and he ended by referring the cause of death to narcotic poison! The facts which subsequently transpired, showed that there could not have been any morphia or opium present in the stomach, that the poison had no existence, and that the woman had died from natural causes. A futile attempt was made to cover this grave mistake by the assertion that some unknown substance resembling opium "in several of its reactions," had in some mode or other found its way into the mixture! The tests for morphia were therefore charged with being open to some unknown additional fallacy, when in reality they had been used in an improper manner, and an incorrect inference drawn from the results. Such mistakes must always occur to the damage of scientific evidence generally, when an undue reliance is placed upon the action of a single test on an unknown organic liquid. Unless morphia or meconic acid, or both, have been separated and obtained from the suspected liquid, no man is justified in swearing that opium is present in a well-marked quantity, and still less that it has been the cause of death! The correspondence of Mr. Davey and Dr. Letheby on this subject conveys an important lesson to medical witnesses, and especially to the "experts" in the chemical branch of medical jurisprudence. (See Med. Times and Gazette, 1857; Aug. 29, p. 229; Oct. 17, p. 407; Nov. 28, p. 560; Dec. 19, p. 641; and Dec. 26, pp. 663 and 673. Also Guy's Hospital Reports, Oct. 1857, p. 496.) It was fortunate in the above case, that some of the medicine taken by the deceased, shortly before her death, remained; and the mistake in the chemical analysis of the contents of the stomach could be thereby clearly demonstrated. Supposing that none of this medicine could have been obtained,—as the husband had administered a dose of it to his wife shortly before her death,—he might have been charged with murder, or the druggist who compounded it,—with manslaughter! The apoplectic symptoms under which the woman died, resembled those of poisoning by opium, and this fact, with the oath of the analytic chemist as to the positive discovery of a grain of opium in the stomach, might have led to a conviction!

In the case of Major Forester (p. 526), on whose body an inquest was held in 1852 (Legal Examiner, Oct. 9, 1852), an "analytical chemist" who gave evidence, deposed to the presence of morphia in the rectum of the deceased, as well as in the urine and the blood, on the ground chiefly that sulphuric acid, and bichromate of potash produced a green color! He appears to have been utterly ignorant that sugar and a large number of organic substances will produce a similar color by setting free oxide of chromium when mixed with sulphuric acid and bichromate of potash; and on this fallacious mode of testing, he confidently swore that he found the two-thousandth part of a grain of morphia in the blood!

It is a remarkable fact that common *saliva* contains a substance which, like morphia, decomposes iodic acid (and produces a blue color with a solution of starch), while at the same time it gives a deep red color resembling that produced by a persalt of iron in meconic acid. This is owing to the presence of an alkaline sulphocyanide. This source of error was first brought to the knowledge of the profession by the evidence given at a trial in Edinburgh (Case of Stewart, 1829), in which the late Dr. Ure was cross-examined for the accused. The means of obviating this error have been elsewhere stated (see p. 552).

Absorbed morphia in the tissues, blood, and secretions.—There is no doubt that morphia is absorbed and eliminated (see p. 73), and according to some chemists it remains for a certain time deposited in the soft organs from which it may be separated. Dr. Vassal thinks, and it is not improbable, that morphia, on becoming absorbed, may undergo some chemical or physical change, so as to be no longer separable from the blood, secretions, or tissues, in a crystallizable state. (Vassal, Considérations Médico-Chimiques, &c. p. 97.) This opinion is partly confirmed by the observations of Flandin. According to this experimentalist, the poisonous salt of morphia is partly changed and neutralized in contact with the living animal fluids, but, according to the dose, traces of it may be still found either in the alimentary canal or in the circulation. He states that he has thus detected it in the fæces, urine, and the tissues of the soft organs; and he holds the opinion that that portion of the poison which kills, remains as such in the organs, and that it is in the power of chemistry to separate it. Admitting that M. Flandin's views are confirmed by the researches of others, it is obvious that the conditions required for the detection of this poison in the tissues or liquids, can rarely exist. When given in small doses, and it proves fatal, it will be difficult to detect it in the soft organs by any chemical processes at present known. When so much of the poison has been taken as to be more than sufficient to destroy life, and a portion of this surplus quantity remains in the stomach or intestines, the analyst may have the hope of detecting it, but so far as I know, it has never been clearly or unequivocally separated from the liver or other organs by any English chemist of repute. M. Stas states that in a case of poisoning at Bruges, in 1845, he detected morphia (free and absorbed) in "all the organs" of the deceased after the body had been buried thirteen months. Again, in 1847 he detected morphia in "the organs" of a man named Bureau, who had died from the effects of arsenic. (Flandin des Poisons, ii, 133.) Neither the quantity detected, the process pursued, nor the organs in which the morphia was actually found, are described in detail, and with respect to the last case, the terms used are so vague that the statement may simply refer to the discovery of morphia in the stomach and bowels.

The process for detecting morphia in the liver, would not differ from that adopted for the stomach and organic liquids. The organ should be sliced in small pieces, and hydrochloric or phosphoric acid in water used as the solvent (p. 557). I have examined the livers and tissues in several cases, in which opium had been taken, and although a liquid or a residue has been obtained, which had a bitter taste, which was reddened by nitric acid, which decomposed iodic acid, and was turned green by bichromate of potash and sulphuric acid, still no alkaloid could be procured; and one or more reactions similar to those above-mentioned may be obtained by an analysis of the livers of persons who have not taken opium or morphia. Either morphia may not be permanently deposited in the organs or it may be changed in its chemical properties before it reaches them (p. 86).

Quantitative analysis.—There are no satisfactory means of determining the *quantity of opium* present in a suspected liquid. Dr. Ure has recommended that we should rely upon the depth of color produced on the addition of sesquichloride of iron to the liquid,—considering that the intensity of the red color will indicate the presence of a large quantity of meconic acid, and consequently of meconate of morphia. The analyses of Mulder have shown that this method is not accurate. The meconic acid does not bear any constant proportion to the morphia in opium. The only plan is to extract the morphia and weigh it.

Proportion of opium in opiate preparations.—*Confection of opium* contains one grain of opium in thirty-six grains. The dose for an adult is from ten to thirty grains. *Compound soap pill.*—Five grains contain one grain of opium. Dose three to ten grains. *Compound pills of storax.*—The strength and dose are the same as in the compound soap pill. *Compound chalk powder with opium.*—Forty grains contain one of opium. Dose five to thirty grains. *Compound powder of kino.*—Twenty grains contain one of opium. Dose five to twenty grains. *Extract of opium.*—Dose one-quarter of a grain to three or four grains. *Wine of opium.*—This is said to have the same strength as the tincture, *i. e.* one grain of opium in twelve or nineteen drops. Dose ten drops to one drachm. *External applications.*—*Liniment of opium* contains one drachm of tincture in one ounce and a half. *Enema of opium* contains thirty drops of the tincture in four ounces. (For *Tincture of opium*, see p. 518.)

CHAPTER XXXVI.

PRUSSIC ACID—EFFECTS OF THE VAPOR—SYMPTOMS—PRUSSIC ACID AS A LIQUID—TASTE AND ODOR OF THE POISON—PERIOD AT WHICH SYMPTOMS COMMENCE—LOSS OF CONSCIOUSNESS AND VOLITION—EFFECTS CONTRASTED WITH THOSE OF OPIUM—CHRONIC POISONING—ACCUMULATIVE PROPERTIES—EXTERNAL APPLICATION—APPEARANCES AFTER DEATH—LOSS OF ODOR IN THE DEAD BODY—FATAL DOSE—TREATMENT.

General remarks.—HYDROCYANIC or PRUSSIC ACID, owing to its rapid and unerring effects when taken even in comparatively small doses, is one of the most formidable poisons with which we are acquainted. The pure or anhydrous acid requires no notice here; since it is not likely to be met with out of a chemical laboratory. The common acid is a mixture of this pure acid with water, and sometimes with alcohol. As it is sold in shops, it varies considerably in strength. I have found different specimens to contain from 1·3 to 6·5 per cent. of the strong acid; but two varieties are now commonly met with—1. The Prussic acid of the London Pharmacopœia, containing about two per cent. (Phillips.) 2. Scheele's acid, containing from four to five per cent. In a case of poisoning which I was required to investigate in July, 1847, the acid which was sold for Scheele's was found to contain only *two* per cent.! (Med. Gaz. xl, 171.) In another instance there was the same deficiency of strength. In recently examining a sample supplied by a druggist as of genuine strength, and with printed precautions as to the dose, I found it to contain only 3·2 per cent. of anhydrous acid. The pharmacopœial acid is found to vary from one and a half to two per cent. These facts are of importance in reference to fatal doses, and the identity of an acid which is alleged to have caused death. In short, there is no certainty respecting the strength of any two specimens sold as the pharmacopœial or Scheele's acid, a subject which requires the serious consideration of medical practitioners who prescribe it. The *medicinal dose* of Scheele's acid is from a minim to two minims,—of the London pharmacopœial acid, from three to five minims gradually increased. The price at which the acid is sold to the public is about two shillings an ounce. On the continent, the acid is met with of a strength rising as high as from ten to twenty-five per cent. The following should be the percentage strength in anhydrous acid of the different varieties of this acid, British and foreign, in *aqueous* solution. Acid of Schrader (acid of the Prussian Pharmacopœia), 1: Dublin Pharmacopœia, 1·6 to 2·82 (Donovan): London and United States Pharmacopœia, 2: Göbel, 2·5: Edinburgh Pharmacopœia, 3·2: Vauquelin and Giese, 3·3: Scheele, 4 to 5: Ittner, 10: Robiquet, 50. Among the *alcoholic* solutions of the acid,—Schrader, 1·5: Bavarian Pharmacopœia, 4: Duflos, 9: Pfaff, 10: Keller, 25 per cent.

Poisoning by Prussic acid is frequently the result of suicide or accident. In 1837–8 there were twenty-seven cases of poisoning by this liquid, nearly all of which were the result of suicide. Of late years it has, however, acquired a fatal celebrity as a means of murder! Several murders have been perpetrated and several attempted by this poison. Among the more recent cases (Reg. v. Bell, York Lent Ass. 1857), the prussic acid was mixed with and sent in a bottle of sherry to the prosecutor!

Prussic acid vapor.—The vapor of anhydrous prussic acid, if respired, would prove almost instantaneously mortal. Even the vapor of the diluted acid accidentally respired may occasion serious symptoms. A medical practitioner, while showing to some friends the effects of Scheele's prussic acid on an animal, accidentally allowed a quantity of the acid to fall upon the dress of a lady who was standing before a fire. The poison was rapidly evaporated, and the lady was immediately seized with dizziness, stupor, inability to stand, and faintness. The pulse was feeble and irregular. Brandy was administered, cold affusion employed, and the patient was exposed to a free current of air. In ten minutes the pulse began to improve, and with the exception of trembling in the limbs, the unpleasant symptoms disappeared. I have known headache and giddiness produced by the vapor from the small quantities used in ordinary chemical experiments. Some caution is required even in smelling a bottle containing a strong specimen of this acid. Chemical experiments show that this poison is always in the act of escaping from liquids which contain it; and the quantity evolved and diffused depends on temperature and the surface exposed to air.

I am not aware that there is any well-authenticated case of death having been caused by the vapor. The celebrated Scheele died suddenly while making his researches on this poison, and it is alleged that he was killed by breathing the vapor of the diluted acid. In October, 1847, a question arose at an inquest in this metropolis, whether the vapor of Scheele's acid had caused death. The deceased entered a druggist's shop, and requested to be shown a bottle of Scheele's prussic acid. He suddenly attempted to snatch the bottle from the hand of the assistant: a struggle ensued, during which a portion of the acid was spilled over the deceased's face, and over the coat of the assistant. The deceased ran into a neighboring shop, and died in about a quarter of an hour. At the inquest it was alleged that death had been caused by the vapor, owing to the acid having been spilled over the deceased's face. Of this, however, there was no proof, as the body was not inspected for the inquest! It is most probable that the deceased had swallowed a sufficient quantity of the acid to cause death.

Dr. Regnauld has reported a case in which a student nearly lost his life by respiring the vapor of prussic acid, as it escaped from a flask in which he was preparing the poison. He lay in a perfectly insensible state for many hours. There was slight lividity of the face, the eyelids were closed, and the pupils were widely dilated: the breathing was difficult, and took place at intervals. The limbs were cold and the pulse was barely perceptible. The muscles of the arms and legs were firmly contracted, but there were no tetanic convulsions. The patient was in a state of complete coma, and could not be roused. After some hours, recovery took place, but the patient suffered from headache and other symptoms. (Ann. d'Hyg. 1852, i, 455.) From these facts there is no doubt that the concentrated vapor would speedily destroy life.

Dr. Chanet has directed attention to the effects slowly produced by prussic acid vapor, upon those who breathe it when diffused in a very diluted state. The process of galvanic gilding and silvering is now very common. Cyanide of potassium is used as a solvent for the metals, and as the solution is freely exposed to the air, prussic acid is always passing off in vapor from its surface. The evolution of the vapor is aided by warmth, and its noxious effects are aggravated by the closeness and want of ventilation in the rooms in which the process is carried on. The whole manufactory is perceptibly infected with the odor, and the workmen are thus compelled to breathe a poisonous atmosphere for many hours together. Dr. Chanet satisfied himself respecting the diffusion of the acid, by placing above the cyanide-bath, a watch-glass containing a solution of nitrate of silver. A white film of cyanide of silver was immediately produced on the surface. Some of the men are speedily obliged to abandon the work, from the feeling of illness produced. The symptoms among those who remain for a long time exposed to the vapor are: dull headache, accompanied by shooting pains in the forehead, noises in the ears, giddiness, dizziness, and other effects indicative of cerebral congestion. Then follow difficult respiration, pain in the region of the heart, sense of suffocation, constriction in the throat, and palpitation, with alternate fits of wakefulness and somnolency. (Gazette des Hôpitaux, 24 Juillet, 1847.) In trying some experiments on galvanic gilding, a few years since, I found that the evolution of the prussic acid vapor was so manifest that nitrate of silver was whitened when exposed in the apartment at some distance, and the whole apparatus was therefore kept covered over.

From the researches of M. Gaultier de Claubry, it would appear that prussic acid is evolved in vapor to a dangerous extent in other manufactures. Some years ago, a patent was taken out in France for the purpose of recovering the alcohol left in the residue of the preparation of the fulminate of mercury. Nitric acid and alcohol are here employed; and in addition to formic acid and hyponitrous ether, prussic acid is a product. The proportion varies according to the degree of concentration of the liquids, but it is sometimes considerable. In saturating the residuary liquid with chalk, prussic acid escapes in vapor, but the odor is in great part concealed by that of the ether. In distilling the liquid, prussic acid was abundantly obtained. On one occasion, the workman who stirred the chalk into the liquid, suddenly felt a severe pain in the head, his strength immediately failed him, and he fell down in a state of unconsciousness; a man who went to assist him was similarly affected. The experiment was performed in the presence of M. de Claubry, and he satisfied himself that these effects were due to prussic acid vapor, the odor of which was distinctly recognized. From only one drop of a portion of liquid distilled by himself, M. de Claubry suffered under the most alarming symptoms, from which he recovered after some hours. (Ann. d'Hyg. 1839, ii, 350.) The free and incautious use of the solution of cyanide of potassium in photography is attended with similar results.

Symptoms.—*Prussic acid as a liquid.*—When a large dose has been taken, as from half an ounce to an ounce of Scheele's acid, the symptoms may commence during the act of swallowing or within a few seconds. It is rare that their appearance is delayed beyond *one or two minutes.* When the patient has been seen at this period, he has been perfectly insensible, the eyes fixed, prominent and glistening, the pupils dilated and unaffected by light, the limbs flaccid, the jaws fixed, frothing at the mouth, the skin cold and covered with a clammy perspiration; there is convulsive respiration at long intervals, and the patient appears dead in the intermediate time; the pulse is imperceptible; and involuntary evacuations are occasionally passed. The respiration is slow, deep, gasping, and sometimes heaving, sobbing, or convulsive.

The following case presents a fair example of the effects of this poison in a large and fatal dose. A medical man swallowed seven drachms of the common prussic acid. He survived about four or five minutes, but was quite insensible when discovered, *i. e.* about two minutes after he had taken the poison. He was found lying on the floor, senseless,—there were no convulsions of the limbs or trunk, but a faint flickering motion was observed about the muscles of the lips. The process of respiration appeared to cease entirely for some seconds: it was then performed in convulsive fits, and the act of expiration was remarkably deep, and lasted for a long time. The deceased swallowed the poison in the act of ascending the stairs of his house. Simon mentions a case in which an ounce was taken, and the symptoms were of a similar kind. There was besides, coldness of the hands and feet, and no pulse could be felt. In such cases, *i. e.* where the dose is large, the breath commonly exhales a strong odor of the acid, which may be perceptible in the apartment. Convulsions of the limbs and trunk, with spasmodic closure of the jaws, are usually met with among the symptoms; the finger nails have been found of a livid color, and the hands firmly clenched. The breathing is generally convulsive, but when the coma or insensibility is profound, it is sometimes *stertorous.* This was noticed in a case which occurred to Dr. Christison (Edinburgh Monthly Journal, February, 1850, p. 97.) It was also observed in the case of *Marcooley* (Reg. v. Burroughs, C.C.C., February, 1857.) The deceased in this case swallowed about two drachms of Scheele's acid in some castor oil. He complained while drinking it that it had a bitter taste. He fell on the floor in a few seconds in an unconscious state; and he died in about an hour, without recovering sensibility or consciousness. In fifteen minutes after he had taken the poison, he was seen by a medical man, who found him breathing stertorously and convulsively. The pupils of the eyes were fixed; there were slight convulsions. No odor was perceived. In the cases of the Smithers family (a mother and two sons poisoned by prussic acid, at Croydon, in 1857), it was noticed that the breathing in one case was of a snoring kind.

Stertorous or snoring breathing has not been hitherto recorded by toxicologists as one of the usual symptoms of poisoning by prussic acid. In the inquiry which took place at Rugeley, in January, 1856, respecting the death of Walter Palmer, it was contended that the fact of the deceased having had stertorous breathing was a proof that he had died from apoplexy, and not, as it was alleged, from prussic acid, administered to him by his brother, William Palmer; but the facts here recorded show that such an inference is inadmissible. On the other hand, there were moral circumstances which rendered it highly probable that deceased might have had a dose of prussic acid administered to him shortly before his death, and that he had really died from its effects.

When a small dose (*i. e.* about thirty drops of a weak acid) has been taken, the individual has first experienced weight and pain in the head, with confusion of intellect, giddiness, nausea, a quick pulse, loss of muscular power, shortness of breathing, and palpitation; these symptoms are sometimes slow in appearing. An increased flow of saliva, as well as vomiting, has been occasionally among the symptoms, when the acid was much diluted, but it is more common to find foaming or frothing at the mouth, with suffusion or a bloated appearance of the face and prominence of the eyes. If death result, this may be preceded by tetanic spasms, opisthotonos, and involuntary evacuations. Vomiting is sometimes the precursor of recovery. (See case, Med. Gaz. xxxvi, 103.) For an account of the symptoms produced by comparatively small doses, see cases by Mr. Hicks (Med. Gaz. xxxv, 893), and by Mr. Pooley (ib. p. 859.) A case which occurred to Mr. Bishop was remarkable in several particulars: the person swallowed, it was supposed, forty minims of an acid (at three and a quarter per cent.) and was able to give some account of his symptoms. He was conscious for a short time after he had taken it, and he recollected experiencing the sensation of his jaws becoming gradually stiff and tight. (Prov. Med. and Surg. Jour. Aug. 13, 1845, p. 516.)

In an accident which occurred in Paris, seven epileptic patients in the Bicêtre were poisoned by an overdose of prussic acid administered in syrup. The quantity actually taken could not be satisfactorily determined. The symptoms, as they are described by Orfila (Op. cit. ii, 286) and Devergie (Op. cit. ii, 281), were as follows: Seven minutes after the poison had been swallowed, all the patients were found lying on their beds in a state of insensibility; they all had convulsions. The respiration was loud and hurried, the mouth covered with froth, the body in a state of perspiration, and the pulse frequent. To this general excitement, there gradually succeeded a mortal collapse. The act of respiration took place at greater intervals, and was of longer duration: the pulse became weak and less frequent, there was cold perspiration, with coldness of the extremities, followed by death. In some, the skin of the head and face was strongly congested, in others it was pale; the pupils were moderately dilated; there was no vomiting; one of the patients made violent efforts to vomit a short time before he died. An attempt was made to put the feet in warm water, but most of them died before this treatment could be adopted. One who survived the longest, and who was thus treated, was shortly afterwards seized with violent convulsions. He evidently felt the warmth of the water, for just before the convulsions came on his countenance expressed much suffering, and his breathing was more hurried. The face, eyes, and head, became also strongly congested. A vein was opened, from which a small quantity of dark liquid blood escaped, but the patient speedily died. The first man died in from fifteen to twenty minutes, the last died in three-quarters of an hour after having taken the poison. Böcker considers that there are three stages in the symptoms when the dose of the diluted acid is small, but, at the same time, sufficient to destroy life: 1. A sense of constriction in the chest,—convulsive breathing with distortion of the features,—giddiness,—the eyes fixed, projecting and staring,—oppression at the heart: 2. Convulsions with opisthotonos,—spasm of the larynx and of the urinary bladder,—loud cries,—involuntary discharge of urine and fæces,—loss of consciousness: 3. General paralysis,—pulselessness,—coma,—relaxation of the muscles,—gradual cessation of respiration and pulsation of the heart,—greatly dilated pupils,—flow of frothy saliva, and death in from half an hour to one hour (Vergiftungen, 1857, p. 23). These stages have no doubt been chiefly based on the results of experiments on animals. In man the symptoms follow each other with great rapidity, or the third stage rapidly supervenes on the first.

It has been erroneously stated that those who took this poison uttered a shriek, or scream, and after this utterance they were incapable of any act of consciousness or volition. At the trial of Tawell (Bucks Lent Ass. 1845), this was poetically described by the counsel in defence, as "the death-scream;" as if it were a uniform or even a common accompaniment of poisoning by prussic acid; and it was contended that as no scream was heard in that case the deceased could not have died from prussic acid! This erroneous opinion appears to have been based on the effects occasionally produced by the poison on animals: it is not in any way supported by observations on man.

The evidence derivable from the *taste* and *odor* of this poison is, in some instances, of importance. The taste is described by Dr. Christison as pungent; some state that it is hot, others that it is bitter. (Pereira.) In one fatal case (December, 1856) deceased complained, on swallowing about two drachms of the poison by mistake, that it had a *bitter* taste. When the common acid is mixed with organic liquids, a taste is not likely to be perceptible unless the dose is large.

With regard to the *odor*, Dr. Christison states that when diffused, it has a distant resemblance to that of bitter almonds; but it is accompanied with a peculiar impression of acridity on the nostrils and back of the throat. (Op. cit. 752.) Orfila also says that it is similar to that of bitter almonds: this is, indeed the common impression. There is, however, a difference between these odors; but the difference is not perceptible to the senses of all, and the only practical point requiring notice is, that the *diluted* odor of bitter almonds would probably be pronounced by many persons to indicate the presence of prussic acid, especially if there existed any suspicion of violent death. Even experienced medical men have to my knowledge been deceived on this point. There are some who are unable to perceive the odor of prussic acid when it exists in large proportion, whether mixed with water or other liquids; while others, again, are peculiarly susceptible of it. With some, it does not affect the olfactory nerves at all, but produces merely a sense of constriction in the throat. These facts appear to me to explain,—why, on being called to a case of poisoning by this acid, or during the examination of a body, some medical men perceive the odor while others do not. When many have to form a judgment on this subject, it is much more common to find disagreement than unanimity. In a case seen by Dr. Christison, in which a man had swallowed a large dose of prussic acid, there was no odor in the breath, near the patient, nor in any part of the room. Two persons could not perceive any odor in six ounces of the warm fluid freshly drawn from the stomach by the pump. (Ed. Month. Journal, Feb. 1850, p. 97.) On other occasions, the odor may be concealed by peppermint, tobacco, or other odors. In a case communicated to me in May, 1850, by Mr. Rake, a man swallowed a large dose of prussic acid, and was observed walking and smoking a pipe. He was found dead in a privy very shortly afterwards; but although the body was still warm, the smell of tobacco-smoke from the mouth completely overpowered and concealed the odor of prussic acid. On opening the body, the smell of the acid was at once perceptible.

Period at which the Symptoms commence. Power of Volition and Locomotion.—One of the most marked effects of prussic acid is to produce insensibility, and loss of muscular power, much more speedily than any other poison. In some instances, there may be loss of consciousness in a few seconds; in others, certain acts indicative of volition and locomotion may be performed, although requiring for their performance several minutes. This is one of the most important questions connected with death by prussic acid. In treating of this subject, Dr. Lonsdale says, that a drachm of Scheele's acid would affect an ordinary adult within a *minute;* and if the dose were three or four drachms, it would exert its influence within ten or fifteen seconds. When the acid is stronger and the quantity larger, we are pretty certain of its immediate action, and the consequent annihilation of the sensorial functions. (Ed. Med. and Surg. Jour. li, 50.) Mr. Nunneley found that in some instances the action of the poison was so expeditious as to prevent the least exhibition of voluntary motion: but in the majority of dogs about *twenty seconds* elapsed before any symptoms were manifested. (Prov. Trans. N. S. iii, p. 75.) Dr. Gerecke gave a teaspoonful of concentrated prussic acid to a doe; symptoms were *instantaneously* produced, and in three seconds the animal was dead (Casper's Wochenschrift, 26 Sept. 1846, 615.)

In his evidence in Rex v. Freeman, Leicester Spring Assizes, 1829, Mr. Macaulay stated that, in one experiment, a dog was killed in three seconds, and the late Dr. A. Thomson observed that a dog had been killed in two seconds. Dr. Christison ascertained that a quantity of poison equivalent to two scruples of medicinal acid did not begin to act on a rabbit for *twenty seconds*, and for so small an animal, two scruples were as large a dose as *five drachms* given to a grown-up girl. (Op. cit. 757.) These very different results appear to me to show clearly that experiments on animals cannot enable us to give a satisfactory solution of this question. We should rather trust to observations made on the human subject. A case was communicated to me, by one of my pupils, where a man was found dead on the seat of a water-closet: he had died from prussic acid, and the bottle which had contained the poison was in his pocket, corked. Many similar facts are recorded which show that while, as a general rule, insensibility may supervene from a large dose of this poison in a few seconds, a person may occasionally retain a power of performing certain acts indicative of consciousness, volition, and locomotion. In a case reported by Mr. Nunneley, a man was enabled to speak rationally, and answer a question, after he had swallowed a fatal dose. (Prov. Med. Jour. July 23, 1845, p. 463, also ante, p. 566.) In Mr. Burman's case (p. 660), sensibility, consciousness, and a power of swallowing, were retained for *two minutes* after a large dose of prussic acid had been swallowed. The importance of this question may be judged of by its bearing on the following case (Rex v. Freeman), which was tried at the Leicester Spring Assizes, 1829. (Medical Gazette, vol. viii, p. 759.) A young man, named Freeman, was charged with the murder of Judith Buswell, by administering to her prussic acid. The deceased was a maid-servant in the family of a druggist, to whom the prisoner acted as assistant. The deceased was one morning found dead in her bed: her death had been evidently caused by prussic acid, and it was presumed that she had taken four and a half drachms of the Scheele's acid; the bottle out of which she must have drunk it, or had it administered to her, held an ounce, and it contained when found three and a half drachms. Owing to the position of the body when discovered, and other circumstances connected with it, it was inferred that she could not have taken the poison herself. Her body was lying at length on the bed, the head being a little on one side. The bed-clothes were pulled up straight and smooth, and they came up to her breast; her arms were under the clothes, and crossed over the chest. On turning the clothes aside, the phial which contained the poison was found lying on her right side. It was corked, and there was a piece of white paper round it;—the leather and string which appeared to have gone round the neck of the bottle were in the chamber-vessel. The medical question at the trial was,—Could this quantity of poison have been taken, and the deceased have retained volition and consciousness for a sufficiently long period to have performed these acts herself? Five medical witnesses were examined, and the opinions of four of these were strongly against the *possibility* of the acts having been performed by the deceased. One of the witnesses ascertained, that a dog, to which the same quantity of acid was given as was supposed to have been taken by the deceased, died in about *three seconds.* The medical opinion was founded on experiments of this kind; there were no cases from the human subject, by which it could be supported. All of the acts to which the opinion referred, might be performed in from *five to eight seconds;* and there is nothing to warrant us in supposing that, under the above-named dose, all power would necessarily have ceased before this period of time had elapsed. On the contrary, there are now numerous facts which show that the symptoms from a fatal dose may be often protracted for *several minutes.* The medical opinion was fortunately completely set aside by circumstances, and the prisoner was acquitted. A similar case occurred in Germany, and is quoted by Sobernheim. A young man swallowed four ounces of an acid (of four per cent.), equivalent to eight ounces of the pharmacopœial strength! He was found dead in bed,—the clothes drawn up to his breast, the right arm stretched out straight beneath the clothes, the left bent at the elbow-joint, and on each side of the bed lay an empty two-ounce phial. There was no doubt of this having been an act of suicide.

In this case more than three times as much acid was taken as in that of Buswell, but even here there was time for the performance of similar acts! It is besides much more difficult to understand, how the poison should have been taken out of two phials than out of one.

A few years since I was required to examine a case of suicide by prussic acid, in which the facts were strongly confirmatory of the views here expressed. The deceased swallowed *three drachms* of prussic acid, and was found dead in bed, the clothes being smoothly drawn up to his shoulders, and there was no appearance of disorder about them, nor was there any sign of struggling before death. On a chair at the back of the bed, but close to it, was the phial which had contained the prussic acid with the cork in it. (G. H. Reports, April, 1845.) There could not be the slightest doubt that the deceased had committed suicide, and that, after swallowing the poison, he had retained sufficient sense and power to perform these acts. In August, 1853, a Mr. Harris destroyed himself at Carmarthen, by taking a large dose of prussic acid. He was found lying on his back in bed, with the bed-clothes, which were quite smooth, drawn up to his chin. His hands were clenched and lying upon his chest, his legs were straight, and his face was pale, but otherwise natural. A glass tumbler was found by his side in the bed, and a bottle, corked (which had contained prussic acid), was on its side in the centre of a dressing table by the side of the bed, but beyond the reach of deceased. In a case reported by Mr. Crisp, in which a very large dose had most probably been taken, the bottle which had contained the poison, with the stopper, was found in the chamber-vessel, and this had been pushed some distance under the right side of the bed. (Lancet, September, 1844.)

It has been supposed that under these circumstances of survivorship, the body should always be found convulsed, and the limbs distorted, but this is not borne out by facts. Here are cases of undoubted suicide, in which the body is lying calm and tranquil without any mark of struggling or convulsions: whether convulsions had taken place, or not, is quite immaterial, since there was nothing to indicate that such symptoms had followed the ingestion of the acid. A girl destroyed herself by prussic acid, and the evidence proved, so far as the facts could be proved, that she had swallowed *an ounce* of the acid, recorked the phial, thrust the bottle to a full arm's length between the feather-bed and the mattress,—got into bed, and then drawn the clothes over her body; there appeared to have been no convulsions. One medical man fancied he perceived the odor of prussic acid about the mouth, but another could not perceive it. (Lancet, June 7, 1845, 640.) This case appears to decide the question, *i. e.* that under a large dose without convulsions,—insensibility or loss of consciousness may not come on until after the lapse of a sufficient time for a human being to perform acts which, a few years ago, from experiments on animals, were deemed impossible!

Locomotion and muscular exertion are, of course, compatible with small but fatal doses of this poison. In a case which occurred to Mr. Hicks, a girl sprang from her seat after swallowing a small dose of acid, threw her arms over her head, gasped for breath, and ran forwards about two yards, before she fell. In one, reported by Mr. T. Taylor, the man ran twelve or fourteen paces before he fell, and remained insensible for a space of four hours,—a very long duration for the effects of this poison without causing death. Other cases have shown that prussic acid does not give rise to insensibility and other alarming symptoms so speedily as it was formerly supposed. Mr. Garson, of Stromness, has reported an instance in which a person, for medicinal purposes, took at least a teaspoonful of prussic acid (the strength not mentioned); the symptoms, however, did not come on for a *quarter of an hour*, when the patient was found insensible. He recovered, and stated that that period of time had probably elapsed between the taking of the dose and the commencement of the symptoms, and that he had employed himself in writing during the intermediate period! (Ed. Med. and Surg. Jour. lix, p. 72.) Perhaps one of the most extraordinary instances on record, in this respect, is that related by Mr. Godfrey. A gentleman, æt. 44, swallowed, it was supposed, half an ounce of prussic acid (strength not stated), but certainly a quantity sufficient to destroy life. After taking it from the bottle, he walked ten paces to the top of a flight of stairs, descended the stairs, seventeen in number, and went to a druggist's shop, at forty-five paces' distance, where he had previously bought the poison, entered the shop, and said, in his usual tone of voice, "I want some more of that prussic acid!" He then became insensible, and died in from five to ten minutes after taking the poison. This case is further of interest from the fact, that although it was an instance of slow death, there were no convulsions, there was no odor of prussic acid about the mouth, and the person died, in the presence of several medical men, without any shriek, or any symptom approaching to it, being observed! (Prov. Med. Jour. Sept. 25, 1844.) In two cases which have come to my knowledge, in each of which a specimen of the acid was sent to me for the determination of its strength, this retention of volition and consciousness existed even after full doses of the poison had been swallowed. The first was a case which occurred at Worcester: the deceased must have swallowed *two grains* of anhydrous acid: he conversed and manifested consciousness and volition for at least *two minutes*, and probably some time longer, after the poison had been taken. He died calmly; there were no convulsions. (Med. Gaz. xl, 171.) In the second case, the quantity of anhydrous acid taken by the deceased, a young man, æt. 23, was 2·54 grains. He had swallowed it in his bed-room: he then descended thirty stairs and walked about twenty paces before he became powerless. He was proceeding to open the front door of the house to go out, when he suddenly fell. The only symptoms observed by a person present were, that "he threw his arms about, and made a noise in breathing, fetching it hard: but he very soon became still." When seen by Mr. Lowe a quarter of an hour afterwards, there was no odor of prussic acid about the mouth or the body. (G. H. R. Oct. 1846, p. 490.) There is, I believe, no other instance recorded in which such a series of voluntary acts has been performed, and such a power of locomotion exerted, after so large a dose of this poison had been taken. It suggests, therefore, additional caution: it shows that full allowance must be made for the occurrence of some delay in the accession of insensibility and powerlessness, even when the dose of poison is large! The facts accumulated in reference to this question are now so numerous and well authenticated, that it is never likely to become again a subject of doubt or dispute in a court of law, or to place the life of an accused person in jeopardy.

Its effects contrasted with those of opium.—If we contrast the effects of this poison with those of opium, we shall find the following general differences. In opium, the coma comes on gradually, and is seldom seen until after the lapse of a quarter of an hour: in poisoning by prussic acid, coma is almost instantaneously induced: even in weak doses, insufficient to prove fatal, this symptom is rarely delayed beyond two minutes. Convulsions may be met with in both forms of poisoning, but perhaps more commonly in poisoning by prussic acid. With respect to the occurrence of this symptom, it is a fair question, whether medical jurists have not too readily adopted views from the results of experiments made on animals—not from observations on man: since in very few instances, when the dose of poison has been *large*, has the patient been seen alive. When the dose was small, but still fatal, convulsions have been sometimes observed. In poisoning by opium the pupils are contracted, except in the advanced stage, when they may be found dilated. In poisoning by prussic acid they are commonly dilated. A well marked remission or intermission of the cerebral symptoms has been frequently noticed before death, in poisoning by opium (see ante, p. 523): this has not been witnessed in poisoning by prussic acid,—the symptoms have been observed to progress in severity until death. In poisoning by prussic acid, the case, if fatal, generally terminates in less than an hour: in poisoning by opium, the average period of death is from six to twelve hours. The time at which the symptoms appear after a liquid has been swallowed, their sudden invasion, the almost immediate loss of sensibility, and the odor of the breath would, under ordinary circumstances, suffice to establish a distinction between the effects of the two poisons.

Chronic poisoning. Accumulative properties.—A question has arisen, whether prussic acid is an accumulative poison; *i. e.* whether, after having been taken in small doses and at short intervals without apparent mischief, it may by accumulation in the system suddenly give rise to all the effects of poisoning, either on a repetition of the same dose, or by a slight increase in quantity. Dr. Lonsdale, who had examined the effects of the acid, denies that it possesses this property, on account of its great volatility and rapid diffusion (Ed. Med. and Surg. Jour. li, 49); and its speedy elimination supports this view (p. 72). The period required to remove entirely from the system a medicinal dose has not been determined; but if an interval of more than a day should occur, we may suppose that there would be no danger from the accumulation of the poison. Hence it is only a too frequent repetition of the acid, in innocent doses at short intervals, which is to be dreaded. Mr. Nunneley ascertained, by his experiments, that when one dose not sufficient to destroy life had been given to an animal, a second and smaller dose, which by itself would not have killed, caused immediately violent symptoms and speedy death. There is one case reported which renders the existence of an accumulative property in the acid, to this limited extent, highly probable; and another has been communicated to me, which also bears out this view. The question is of some importance in respect to the medicinal use of the acid; for serious effects have repeatedly resulted from a slight increase of the dose. The following case has a bearing on this question, and is of some interest, as it involved a respectable English physician, practising at Nice, in a charge of malapraxis. Dr. Gurney was called to a young lady who had been for some weeks in ill health, and was at the time suffering from severe spasmodic pain in the abdomen. He prescribed muriate of morphia and prussic acid: a sixth of a grain of the morphia, and a drop and a third of prussic acid (of 1·5 per cent.) at each dose. Fifteen doses were given throughout the day, making a total quantity of two grains and a half of morphia and twenty drops of diluted (= 3-10ths of a grain of anhydrous) prussic acid. The last dose was given fifteen hours before death. The patient at no time lost her consciousness or sensibility, nor did she suffer from any symptom indicative of narcotic poisoning. The whole of the prussic acid given at once would probably not have destroyed life, and although the total quantity of the muriate of morphia might (if given at once) have proved fatal, it was impossible to refer death to it in the absence of any one symptom indicative of poisonous effects. She answered questions and was quite sensible until within an hour and a half before her death, although thirteen hours had elapsed since the last dose was taken! (Med. Gaz. xxxix, 905.) The inspection revealed a sufficient cause of natural death, in disease of the liver and gall-bladder. Dr. Gurney was thrown into prison on a charge of some informality. The facts of the case were referred to Dr. Babington, Mr. Cooper, and myself: we agreed in certifying that there was not the least ground for imputing poisoning, and that the death arose from natural causes. Dr. Gurney was subsequently liberated.

Effects of external application.—Prussic acid is said to act through a wounded portion of skin. Sobernheim mentions the case of an apothecary at Vienna, who died in an hour from the entrance of the poison into a wound in the hand, produced by the breaking of a glass vessel in which it was contained. It is also said to act through the unbroken skin; but this certainly does not appear to be the case with the common diluted acid. The acid would doubtless produce all the effects of poisoning, if applied to an ulcerated or any highly absorbing surface. It has been found, in experiments on animals, that the poison acts with the same rapidity and certainty on applying it to the mucous membrane of the conjunctiva, rectum, or vagina, as when swallowed. (Prov. Trans. N. S. iii, 84.) Dr. Christison states that three drops of concentrated acid projected into the eye of a cat, acted on it in twenty seconds, and killed it in twenty more; and the same quantity dropped on a fresh wound in the loins acted in forty-five, and proved fatal in one hundred and five seconds. (Op. cit. 757.) These facts will suggest a caution in reference to the medicinal employment of prussic acid locally. It is sometimes used, and with good effect, to allay the violent itching which attends certain diseases of the skin. It has also been applied to the neck of the uterus for the alleviation of pain. As, in the latter case, it meets a highly absorbing surface, care should be taken so to regulate the proportion of acid, both in absolute quantity and degree of dilution, as to prevent the possibility of danger. Dilution with water does not appear to make so much difference in the effects as absolute quantity, and this may be regulated by the known fatal dose.

APPEARANCES AFTER DEATH.—The body often exhales the odor of prussic acid when seen soon after death; but if the dose of poison has been small, or if the body has remained exposed for some hours before it is seen, and especially if it has been exposed to the open air or in a shower of rain, the odor may not be perceptible. In a case in which a person poisoned himself with two ounces of the acid, and his body was examined twenty-eight hours after death, the vapor of prussic acid which escaped on opening the stomach was so powerful, that the inspectors were seized with dizziness, and obliged to quit the room hastily. This may serve as a caution in conducting an examination. In cases of suicide or accident, the vessel or vessels out of which the poison has been taken will commonly be found near; but there is nothing to preclude the possibility of a person throwing it from him in the last act of life, or even concealing it, if the symptoms should be protracted. These vessels should be examined for the odor when the body is first seen. Putrefaction is said to be accelerated in these cases; but, from what I have been enabled to collect, there seems to be no ground for this opinion, any more than in a case of poisoning by opium. Orfila has shown that in most instances of sudden death from whatever cause, putrefaction is, *cæteris paribus*, accelerated; and the fact that in a few cases of death from prussic acid, the bodies have speedily putrefied, has improperly led to this condition being set down as one of the characters of poisoning by the acid.

The following appearances are met with in the body. *Externally*, the skin is commonly livid, or is tinged of a violet-color; the nails are blue, the fingers clenched, and the toes contracted; the jaws firmly closed, with foam about the mouth; the face bloated and swollen; and the eyes have been observed to be glassy, very prominent and glistening, and the pupils dilated; but this condition of the eyes exists in other kinds of death. *Internally*, the venous system, and the liver, spleen, and kidneys are congested with dark-colored liquid blood: the *stomach* and alimentary canal are in their natural state; but in several instances the stomach has been found strongly reddened and congested. The mucous membrane of the stomach of a dog which died in a few minutes from a dose of three drachms of Scheele's acid, was intensely reddened throughout, presenting the appearance met with in cases of arsenical poisoning. In a large number of experiments upon dogs, Mr. Nunneley, states that he found generally a congested condition of the mucous membrane of the stomach: if empty at the time the poison was taken, the organ was much contracted, and of a brick-red color. The same appearance of congestion was observed on the mucous membrane of the vagina, the rectum, and conjunctiva, when the acid was applied to those parts. (Prov. Trans. N. S. iii, p. 79.) Redness of the stomach was noticed in the cases of the Parisian epileptics (Ann. d'Hyg. 1829, i, 507); and Dr. Geoghegan, of Dublin, observed redness of the stomach in the case of a healthy man, æt. 30, who was found dead from a large dose of prussic acid. The body was inspected five hours afterwards: rigidity had commenced, but there was some warmth. The face was pale, the eyes were half-closed, not presenting any remarkable brilliancy or prominence, nor was there much dilatation of the pupils. The mouth was closed, and no froth issued from it. The abdomen was the only cavity examined. The muscles were red, and gave out, on section, much fluid blood, which had a strong odor of prussic acid; the odor of the poison was also perceptible in the abdomen. About eight ounces of a thick farinaceous mass were found in the stomach; the odor of prussic acid was very perceptible in this organ, but it was mixed with that of rancid food. The mucous membrane had everywhere, except at the splenic end and posterior wall, a vivid inflammatory redness, of a well-marked character, and it was, to a considerable extent, lined with a layer of viscid mucus. The coats were not thickened, but the submucous and peritoneal coats were decidedly red. The posterior wall, at the splenic end, was of a chocolate color, with spots of effused blood: the great venous trunks stood out in relief as dark blue lines. The mucous membrane, even when washed three times in water, gave out a strong odor of prussic acid. In a case which I examined in May, 1850, in which death had been caused by a large dose of the acid, there was also a general redness of the stomach.

The odor of the poison, if not observed in the body, is generally perceptible in the stomach for several days after death, unless the quantity of poison is small, and it is mixed up with other strongly smelling substances (p. 580). If death has been rapid, the dose large, and the inspection recent, as in the case just related, all the cavities as well as the blood have the odor. Besides these appearances, the brain and lungs have been found congested, although not invariably. The *blood* is, in some instances, quite liquid, in others, thick and semi-coagulated. (Heller's Archiv. i, ii, 1845, p. 143.) In most cases this liquid has been found of a very dark color,—in a few, red (Heller's case, supra), and in other cases again of a violet or pinkish hue. Heller found, by a chemical and microscopical examination, that in one instance the blood contained no fibrin (supra). In two instances reported by Mertzdorff, the contents of the gall-bladder had a blue tint; but this appearance may have been owing to accidental causes, as, in the generality of cases, there has not been observed any abnormal change in the bile. The air-passages are said to have been found reddened; but it is not impossible that this redness may have depended on other causes. Death commonly takes place with such rapidity, as scarcely to allow of the production of any well-marked morbid changes in the body. Dr. Geoghegan met with a case in which a man swallowed an ounce of prussic acid, and was found dead, and the only morbid appearance of note discovered was a patch of dark red extravasation under the mucous membrane of the stomach near the pylorus. The stomach, in this case, exhaled the odor of hydrocyanic acid, although it had been exposed for *three days*, and the poison was easily detected, in its contents, by the usual processes. In a case reported by Mr. Pooley, a dark color of the blood appears to have been the only striking appearance (Med. Gaz. xxxv, 859): in this instance the lungs were not congested; in a case by Mr. Hicks they were much congested (Med. Gaz. xxxvi, 460); while in another case they were only partially congested. (Prov. Med. Jour. July 30, 1845.) In one reported by Mr. Crisp (Lancet, Sept. 14, 1844), the abdominal and thoracic viscera were healthy, with the exception that they had a purple color from the blood: he could perceive no odor of the poison. Casper found, in another case, the liver, kidneys, and the large veins of the abdomen and chest, as well as the lungs, filled with dark fluid blood; there was a small quantity in the right ventricle, while the brain and its membranes were bloodless. There were red patches on the stomach and œsophagus (Casper, Ger. Med. i, p. 432). Congestion of the brain and its membranes, of the lungs, heart, mucous membrane of the stomach and bowels, of the liver, spleen, and kidneys, have been found more or less in different cases, but there is no constancy or uniformity in these appearances; and but for the presence of the well-known odor of the poison, there would have been no suspicion of the cause of death. In some instances this odor is not perceptible; hence an inspection of the body can throw but little light upon the case in an absence of a knowledge of symptoms and of chemical evidence. In a case which occurred to Mr. Ilott, in which probably death had been occasioned by a large dose, there was slight congestion of the cerebral vessels, but the brain was otherwise healthy, and there was no effusion. The lungs were congested, and the large veins of the chest filled with blood; the lining membrane of the gullet was intensely reddened; the stomach in various points, but more especially near the gullet, was inflamed or congested; the mucous coat was softer than usual, but corrugated. There was a distinct smell of prussic acid in the contents. (Med. Times, June 6, 1846, 197.)

In July, 1847, the late Dr. Streeten communicated to me the particulars of a case in which an adult died in five minutes from a dose equivalent to about *two grains* of anhydrous acid. The following appearances were met with: Countenance (particularly the lips) livid; neck, shoulders, and all the back part of the body, purple. On dividing the integuments, dark blood flowed freely; the blood was universally dark and quite fluid; the lungs were gorged with dark blood, which had to a considerable extent gravitated to their posterior portions. The right auricle and right ventricle of the heart, and vena cava, were full of dark fluid blood; the left ventricle was firmly contracted and quite empty. Five out of six gentlemen did not perceive any odor of prussic acid upon approaching the body, either before or after it was opened. The abdominal viscera were healthy; the urinary bladder was half full of urine, which exhaled no unusual odor; the brain was natural, but full of fluid blood. The stomach, particularly at its cardiac extremity, had a very red appearance, and in some of the patches oozing of blood had evidently taken place, while in others the mucous membrane had a brownish color. It contained about one ounce of a raspberry-colored fluid, and had a strong smell of bitter almonds. Traces of prussic acid were discovered in it by distillation.

In Marcooley's case (Dec. 1856, p. 639) the eyes were closed, the pupils dilated, the lips were purple and swollen, the tongue slightly protruded between the teeth, and the skin was generally dark and livid. There was no distortion of the limbs, and the muscles were not very rigid (twenty-four hours after death). A frothy mucus had escaped from the nostrils. The lungs were congested, the heart was healthy, the right auricle contained much dark blood, the left cavities were empty. The blood was universally liquid. The liver was engorged, the kidneys were healthy. The vessels of the stomach were much congested with dark blood, and the smell of prussic acid was strongly perceptible in the contents. The mucous membrane of the stomach was of a dark red color from congestion; the head was not examined.

In a case communicated to me by Mr. Newham, in which a man had died from the effects of three drachms of prussic acid, the following appearances were found: The membranes of the brain were perfectly natural in every respect; the substance of the organ was also firm and natural. The quantity of serous fluid contained in the ventricles was less than usual; but it was strongly impregnated with the odor of prussic acid. The plexus choroides were pale and bloodless. The lungs were healthy; at the upper part, the organs were of a light red color; at the lower part, they were full of a dark-colored muddy-looking blood. The heart was natural, but contained very little blood; this was of a dark and muddy hue, and strongly impregnated with the odor of the poison. The liver was larger than natural, and there were several spots of medullary deposit about it. The gall-bladder was nearly empty, and the bile was dark-colored. The pancreas was healthy, and the spleen, externally, was of a bright violet-purple color. The urinary bladder was natural: it contained no urine. The stomach contained more than half a pint of viscid liquid having a strong odor of prussic acid. A portion of the mucous membrane, at the greater curvature, was of a deep red hue. The intestines were healthy, but generally empty; the mucous membrane of the duodenum was slightly reddened in patches; and, on being laid open, there was a strong odor of the poison. The lower portion of the large intestines was quite empty, the deceased having involuntarily passed the fæces and urine while under the influence of the poison, and probably in the act of dying. (Guy's Hosp. Rep. April, 1845.)

A complete account of the appearances observed in the bodies of the seven patients accidentally poisoned in a Parisian hospital by an overdose of prussic acid, has been published by Orfila. (Ann. d'Hyg. 1829, i, 507, 1841, ii, p. 409, and ante, p. 566.) The inspection was made by MM. Adelon, Marc, and Marjolin. X., æt. 15, was seized with convulsions in about eight or ten minutes after he had taken an overdose of hydrocyanic acid syrup: he then fell into a state of collapse, and died in half an hour. It was observed in this, as well as in the six other cases, that the symptoms were marked by two distinct periods,—one spasmodic or convulsive, indicative of irritation,—the other of collapse or relaxation; exactly like those observed in dogs poisoned by the acid. An inspection of the body was made twenty-four hours after death. There was no mark of violence, excepting a slight redness on the left instep, produced during the convulsive fit; there was lividity of the back; the head, face, and lips were violet, and slightly swollen; a frothy sanguineous liquid issued from the mouth and nose; the mouth was so firmly closed, owing to the general rigidity of the muscles, that it could not be opened; the eyes were closed, and, on separating the lids, the pupils appeared somewhat dilated. On making an incision into the muscles, they were found of their usual color. The interior of the mouth, throat, and gullet presented nothing remarkable. The alimentary canal, being properly secured by ligatures, was removed for the purpose of collecting the contents. Those of the stomach were placed in a vessel, and this organ was then examined; no particular odor was remarked; here and there were patches of redness on the mucous membrane, especially on the folds; towards the intestinal opening the membrane had a granular appearance, owing to the enlargement of the mucous glands, but there was no sign of ecchymosis or erosion. Red patches, due to capillary injection, with enlargement of the mucous glands, were observed in the small intestines. At the lower part of the small intestines, a small quantity of blood was found effused beneath the mucous and muscular coats. The large intestines were healthy, and contained fæcal matter of the ordinary color and consistence. The liver, spleen, and kidneys were natural, excepting that they contained a larger quantity than usual of dark-colored blood. The membrane covering the kidneys was easily detached. The bladder was healthy, and contained urine. The heart appeared natural: there was no change of color, or any alteration in its substance; it was quite empty, containing neither fluid nor coagulated blood. The larger arteries were likewise empty, but the veins, on the contrary, were filled with liquid blood of a dark color. The lungs were of a reddish color anteriorly, and slightly congested posteriorly. The mucous membrane of the air-passages was of a deep red color, and the lower portions of the bronchial tubes contained a frothy sanguineous liquid. On raising the scalp, a moderate quantity of blood escaped. The sinuses of the dura mater were full of dark fluid blood. The substance of the brain presented no mark of inflammation or ecchymosis,—it was softer, and its vessels were more congested than usual. The spinal marrow, examined throughout its whole length, was quite healthy.

The following appearances were observed in a case in which the quantity of poison taken was supposed to have been equal to four ounces of Scheele's acid (twenty grains of anhydrous acid). The deceased, a young man, was found dead in bed. The body was examined thirty hours after death. At this time it was somewhat decomposed, the scrotum being of a livid color, and in places deprived of its cuticle; several livid red spots were observed on the face, about the chest, neck, and shoulders; there were also a few vesicles on the left thigh. The body did not emit any odor of bitter almonds (prussic acid). The pupils were neither dilated nor contracted; the teeth were not closed. The limbs were somewhat flexible, the nails blue, and the fingers bent. The stomach and greater part of the intestines could be readily torn, the former contained a viscid mass having the odor of prussic acid; its internal surface was of a bright red color and streaked with blood, especially in the neighborhood of the two apertures. The mucous membrane of the intestines was also reddened in many places. The liver, spleen, and kidneys were of their natural color, and were loaded with fluid blood of a bluish-black color (schwarz-blau). The bile was of a dark blue color. The bladder and pancreas were both healthy. The muscles generally were of a darker color than natural. The lungs were healthy in structure, but had the same bluish-black color as the blood. The right and left cavities of the heart were filled with fluid blood containing a few coagula. There was no serum in the pericardium or in either pleural cavity. The tongue was not red, but the windpipe and the gullet, as far down as the stomach, were of a bluish-black color, and in the windpipe there was a large quantity of blood. The brain was healthy, but its vessels and sinuses were loaded with blood, so that it streamed forth when the scalp was cut into and removed. (Case by Merzdorff, in Horn's Archiv. für Med. Erfahrung, 1823, ii, p. 55.)

After this description of the appearances met with in death from a large dose of the poison, it may be proper to state those found in the body of an adult female killed by the smallest dose of prussic acid yet known to have destroyed life—nine-tenths of a grain. The inspection was made ninety hours after death. The teeth were clenched, and foam was still adhering round the mouth; the face was of a dusky red hue, and the whole of the depending part of the body of a dark purple or violet color: it had very much the appearance of the body of a person who had died from asphyxia. The dura mater and sinuses were much congested, and the whole of the substance of the brain was dotted with blood, which was fluid and very black; the ventricles were empty, and the plexus choroides pale and bloodless; but no odor of prussic acid was perceptible. On opening the chest, the odor was more plainly perceived than in any other part of the body; the lungs were much congested but otherwise healthy; the right ventricle of the heart was distended with fluid black blood. The stomach contained four ounces of liquid smelling strongly of prussic acid; its lining membrane was healthy, with the exception of a small patch of redness near the cardiac orifice: but as the deceased had suffered from gastric symptoms, this may not have been due to the action of the poison. The liver, gall-bladder, and kidneys were healthy, except that the latter were congested, and had a dark pinkish hue. (Med. Gaz. xxxvi, 460.)

In inspecting the body of a person who has been poisoned by this acid, it is customary to place great reliance on the detection of the *odor*. Nitrobenzine, the essential oil of bitter almonds (free from prussic acid), noyau, and various liqueurs, have an odor which might easily be confounded with that of prussic acid. Assuming that there are none of these sources of error, it may be stated that the odor of prussic acid in a dead body is evidence of the presence of the poison; but the absence of odor is no proof of its absence. In Reg. v. Tawell (see post), Sir F. Kelly was instructed by his chemical advisers to make this fallacious criterion a strong point with the jury, but the attempt to mystify a plain case wholly failed. As the allegation, that when there is no odor there is no poison may be again made, and affect the position of an innocent as well as of a guilty person, it will be proper to consider the conditions under which the odor may not be perceived in the dead body, in the contents of the stomach, or, indeed, in any organic liquid. These may be referred to—1, the smallness of the quantity of acid present; 2, volatilization by long exposure to air; 3, the smallness of the dose taken, and its entire removal by absorption and elimination when the person has survived some time; 4, the degree of dilution of the poison with water or other liquids; and, lastly, its concealment by other odorous bodies, such as various organic liquids, peppermint, or bitter almonds.

(G. H. Reports, April, 1845.) Dr. Geoghegan detected the odor three days after death. Dr. Lonsdale found, in his experiments on animals, that the smell might be perceived for eight or nine days after death, although he could not detect the acid chemically for more than four days. (Ed. Med. and Surg. Journ. li, p. 52.) In the case of Ramus (Ann. d'Hyg. 1833, 365), the odor was detected in the liquid distilled from the stomach *seven* days after death, and it yielded traces of prussic acid: but it was not perceived before distillation. This fact of the detection of the odor *after* but not *before* distillation, well known to all medical jurists, was made a point of unnecessary difficulty in the evidence in Tawell's case, although the simple explanation is, that the poison is thereby separated more or less from other odorous substances which tend to conceal it. From some cases which have occurred since, as well as from experiments on the dead stomach and intestines, I have satisfied myself that the acid may be separated by distillation when the odor is not perceptible.

In one instance in which three drachms had probably been taken, I found no odor in the stomach or its contents after twelve days. (G. H. Reports, April, 1845.) In a case examined by Mr. Hicks, ninety hours after death, where not more than nine-tenths of a grain had been swallowed and the individual died in twenty minutes, the contents of the stomach smelt strongly of prussic acid. It was also perceived on opening the cavity of the chest; but there was no odor of the poison about the mouth or in the room, although Mr. Hicks was present ten minutes before death. (Med. Gaz. xxxv, 893.) In a case examined by Mr. Pooley twenty-two hours after death, the smell was perceived in the stomach, but in no other part. (Ib. p. 859.) In another, communicated to the Lancet (Sept. 14, 1844) by Mr. Crisp, in which a large dose had probably been taken, the odor of prussic acid could not be detected in any part of the body, although the inspection was made only seventy hours after death. In a case in which a man swallowed nine-tenths of a grain and began to recover after four hours, the matters then for the first time thrown off the stomach had no smell of prussic acid. (Med. Gaz. xxxvi, 104.) Neither the dose nor the circumstances under which the body is exposed, will always suffice to explain these anomalies. The poison may probably undergo some change after it is absorbed.

In the stomach of a dog poisoned by Mr. Hicks, three drachms of Scheele's acid having been introduced by the stomach-pump, the odor was perceptible to some persons (but not to others) twenty-four hours after death; although the stomach had been laid quite open, freed of its contents, soaked in cold water, and placed for some time under a current of water. Mr. Hicks and I subsequently detected prussic acid in it by tests, both with and without distillation. (Med. Gaz. xxxvi, 328.) Hence it follows that at the inspection of a body, if recent, there may be no odor of prussic acid, until the cavities are opened, and if after some days, it may not be perceived in the contents of the stomach, although prussic acid may be obtained by distilling them.

A case occurred in France, in 1841, which shows the great danger that may result from inferring the presence of this poison by the supposed odor. A M. Pralet, æt. 64, was taken suddenly ill on the evening of the 13th January, 1841: he became insensible, and died six hours after the attack. The body was buried on the 16th, and exhumed on the 20th of the same month. Three medical men, assisted by a chemist (pharmacien), undertook the investigation. As the result of their inquiries, they came to the conclusion, that M. Pralet had been poisoned by hydrocyanic acid, and M. Heritier, the nephew of the deceased, was charged with the murder of his uncle. It appeared in evidence that the deceased had dined as usual on the 13th of January, and that at eight o'clock in the evening, he ate a small piece of bread and cheese, and drank three or four glasses of white wine. He was in the act of standing, when he took the last glass; and he had scarcely swallowed it, when he tottered, complained of illness, and became insensible. Various remedies were applied: he vomited a quantity of acid vinous liquid, recovered his consciousness, and said he experienced no pain. Nevertheless, his features were sunken, his face was pale, and his tongue and mouth were slightly drawn to the left side. In spite of remedial measures, there was a relapse. He became again unconscious and quite insensible to pain: the mouth was still more drawn on one side, and there was a tetanic stiffness of the left arm. The pulse, which up to this time had been full and regular, became weaker, and the patient expired at two o'clock in the morning, without having had any convulsions before death. The inspection of the body was made seven days after death. There was no putrid odor about it, but the face was discolored. There was lividity of the skin in patches; the subcutaneous veins were filled with black blood, and the nails were blue. On opening the abdomen, there was a peculiar odor, the nature of which could not be exactly specified: it was afterwards thought to be *more like* that of bitter almonds than any other substance! The stomach was collapsed, and the veins injected, especially towards the cardia; the heart and large vessels were empty, the lungs were soft, small, filled with dark blood, and having the same peculiar odor as that found in the abdomen. The surface of the brain was strongly injected with dark blood. On raising the hemisphere, a clot of the size of an egg was found: this had also the peculiar odor. There was a large effusion of blood above the tentorium. The inspectors stated that a greater degree of cerebral congestion could scarcely have existed. An analysis of the contents of the stomach was made by applying tests to the distilled liquid. This liquid had a strong odor (not defined) which resembled that of prussic acid when sulphuric was added to it, and the mixture was diluted with twenty-three times its weight of water! The result of the application of the tests was, that in the opinion of the inspectors, *faint traces* of prussic acid were present. (Annales d'Hyg. 1841, ii, 399, and 1843, i, 103, 474.)

The nephew was tried for the alleged murder, convicted on this medical evidence, and narrowly escaped execution. An appeal was made against the sentence, and the opinion of Orfila in reference to death from poison, based on the facts of the report, became a subject of deliberation for three days. Orfila contended: 1. That there was no proof, either from odor or chemical analysis, that prussic acid was present in the body; 2. That the symptoms and appearances were not like those met with in poisoning by prussic acid, but, on the contrary, that they clearly indicated apoplexy from disease; 3. That death had been caused by apoplexy, and no crime had been committed. The result of this inquiry was, that the accused was discharged as perfectly innocent!

The symptoms in this case were wholly unlike those caused by prussic acid; the partial paralysis, with the occurrence of a remission, showed the great probability of the attack being due not to this poison, but to disease of the brain. The appearances were such as would be found in a case of apoplexy (effusion of blood), and unlike those caused by prussic acid. With respect to the *odor* observed in the body, the inspectors required some time to consider whether it was, or was not, like that of bitter almonds! It could have furnished no evidence in so doubtful a case, unless the poison had been clearly and unequivocally detected. The chemical evidence was, however, not merely defective, but in many respects completely erroneous; the presence of any trace of prussic acid was not even rendered probable by the results. But for a proper medico-legal investigation of the facts, made by Orfila, M. Heritier, the nephew, would probably have been guillotined! This case shows as strikingly as that of Dore and Spry (p. 210), on what a thread the life of an innocent person, unjustly accused of the crime of poisoning, may depend. Courts of law, both in France and England, wisely act upon the principle, *Cuique in suâ arte credendum;* but the above case shows that this confidence may be carried too far, and that special experience and special knowledge are absolutely required for the solution of these important questions.

In Rex v. Donellan (Warwick Lent Ass. 1781), there was no chemical evidence of the nature of the poison, but the odor of bitter almonds was plainly perceived by one witness in the draught administered to the deceased; and this fact, coupled with the symptoms, the rapid death, and the moral circumstances of the case, left no reasonable doubt that prussic acid contained in laurel water was the cause of death. It was a question in Belaney's case (p. 588), how far the odor of prussic acid was likely to be concealed when the poison was mixed with a saline solution (sulphate of magnesia). From experiment, I have not found that this causes any other difference than mere dilution with an equal quantity of water. Prussic acid is constantly and rapidly evolved from all solids and fluids which contain it, but the evolution appears to be slow in proportion to the degree of dilution.

It is owing to this great volatility that a specimen of strong acid exposed for a short time to the air in a bottle without a stopper, or with an imperfect stopper becomes considerably reduced in strength. A few hours' exposure will render it wholly different in strength from the sample from which it was originally taken. This must be remembered in identifying by chemical analysis a sample of prussic acid found near a dead body. When the acid is placed in a jar in a state of mixture with oil, and the jar is tightly secured by bladder, the vapor of the acid rapidly traverses the bladder, and may be easily detected on the outside. The result is the same whether the bladder is wet or dry. Thus the vapor may traverse the coats of the unopened stomach, and diffuse itself through the abdomen and chest; hence, the odor is often plainly perceptible in these cavities. It may even traverse the skin and escape from the interior to the exterior of the body. This extreme volatility tends to account for the disappearance of the poison in the dead body.

Bearing these facts in mind, the viscera, in a case of poisoning by prussic acid, should be placed in a glass vessel with a closely fitting stopper, or in a jar well secured with bladder, and on the outside of this a double layer of tinfoil. If the jar is merely covered with paper, the acid is speedily lost. Its vapor will rapidly traverse several folds of paper.

FATAL DOSE.—In a case of poisoning by this acid, which occurred to Dr. Geoghegan, twenty-seven minims of the English pharmacopœial strength (at two per cent.) were taken by a gentleman without any effect, the medicinal dose having been gradually raised to this point; but no bad consequences whatever had resulted from the acid previously taken. He increased the dose to thirty-six minims, and in two minutes he was seized with the usual symptoms, and nearly lost his life. (Dublin Med. Journ. viii, 308.) The quantity of anhydrous acid (0·66 grain) swallowed in this dose, was less than three-quarters of a grain, *i. e.* about equal to eighteen drops of Scheele at four per cent. Cases hitherto observed show that there is a very narrow line between the quantity of this poison which may be taken with impunity, and that which may produce death. In determining the quantity necessary to prove fatal, we must, it appears to me, for the purpose of legal medicine, reject the results obtained by experiments on animals, and look to those facts only which have been ascertained from numerous observations in the human subject. In general the quantity taken is large; but the *smallest* dose known to have caused death was in a female whose case has been reported by Mr. Hicks. (Med. Gaz. xxxv, 896.) The patient, a healthy woman, died in twenty minutes from a dose equivalent to *nine-tenths* of a grain of anhydrous prussic acid. This was equivalent to *forty-nine* minims of the London pharmacopœial acid, and taking Scheele's acid at four per cent. (Pereira), to about *twenty-five* minims of Scheele. In a case that occurred to Mr. Taylor (Med. Gaz. xxxvi, 104), a stout healthy man swallowed this dose, *i. e.*, nine-tenths of a grain, by mistake, and remained insensible for *four hours*, when he vomited and began to recover. The vomited matters had no odor of the poison, showing that, if not concealed by other odors, the whole of the acid must have been here absorbed (ante, p. 580). He had a very narrow escape of his life. Dr. Banks has published a case in which a female recovered after swallowing thirty minims of prussic acid (Ed. Med. and Sur. Jour. xlviii, p. 44), but the strength of the acid in this instance was not determined.

The *largest* dose from which an adult has recovered, with the exception of a remarkable case recorded by Böcker, was in the case of Mr. Burman (Lancet, Jan. 14, 1854). This gentleman, æt. 60, a man of strong constitution, took by mistake a *drachm* of prussic acid, equivalent to 2·4 grains of anhydrous acid. In a few seconds he perceived his mistake, and swallowed half an ounce of aromatic spirits of ammonia with a little water. He called to his son; told him what had occurred; spoke hurriedly, and breathed deeply. Some solution of green sulphate of iron was given *two minutes* after the poison had been swallowed. Insensibility then came on, and the respiration was deeper and slower.

Four minutes after taking the poison cold affusion was employed, and sulphate of iron and spirits of ammonia were administered. Vomiting with convulsive shuddering took place. In twenty minutes consciousness returned, and in fifteen minutes later he was able to walk up stairs to bed. He perfectly recovered, but in the absence of the treatment resorted to, it is most probable that he would have died.

Dr. Christison has reported in the Edinburgh Monthly Journal (Feb. 1850, p. 97), the case of an adult who recovered after having taken a dose of a grain and a half or two grains of anhydrous acid. The symptoms were such that the man would have died, but for the immediate treatment, which consisted in the evacuation of the stomach by the stomach-pump, and in pouring a current of cold water on the head. It is a remarkable fact that in this case no bottle or vessel could be found in the room or under the window. The patient hastily summoned his wife one evening, told her that he had taken prussic acid, and immediately fell down senseless on a sofa, without either cry or convulsion, but drawing his breath deeply, forcibly, and slowly. He recovered in about three hours, but had an unusual disposition to sleep even on the following day. Another remarkable case of recovery from a dose nearly as large occurred to Mr. Bishop (Prov. Med. Jour. Aug. 13, 1845, p. 517). The person swallowed, it was supposed *forty minims* of an acid at three and a quarter per cent. Taking the minim as equal to the grain, although it may be a little more or less according to circumstances, this is equivalent to about one grain and one-third of anhydrous acid. The man was for a short time conscious, got into bed after taking the poison, and spoke. He felt his jaw become stiff, and then remained insensible until roused by the cold affusion. The fact of recovery having taken place on these occasions, must not lead us to suppose that such large doses could be commonly taken with impunity. If we refer to the chapters on arsenic and corrosive sublimate, we shall find that persons have recovered from doses of these poisons much larger than those which have proved fatal in other cases. The same circumstance is observed in respect to all other poisons. Judging by the effects produced in Dr. Geoghegan's case, from 0·66 grain of anhydrous acid,—from the fact that death took place in Mr. Hicks's case from nine-tenths of a grain, and that, in another instance, a strong adult had a narrow escape of his life from the same dose, we shall not be wrong in assuming that a quantity of Scheele's acid (at five per cent.) *above twenty grains* (*i. e. one grain of anhydrous acid*), or an equivalent portion of any other acid, would commonly suffice to destroy the life of an adult. This I believe to be the nearest approach we can make to the *smallest fatal dose.* It is scarcely necessary to remark that the quantity of poison found in the stomach is the surplus of that which has actually destroyed life. On the trial of John Tawell, the Quaker (Aylesbury Lent Assizes, 1845), it was made a great point in the chemical defence, undertaken by Mr. Herapath and Dr. Letheby, that, as not more than *one grain* of anhydrous prussic acid was separated from the contents of the stomach, and this, in their opinion, was not sufficient to have caused death, *therefore*, the deceased could not have died from the effects of this poison! That an astute lawyer should make a perverted use of scientific evidence for the purpose of obtaining a verdict from a jury, is not surprising; but that persons professing, as "experts," to be acquainted with the action of poisons, should either suggest or allow suggestions to be made, the only tendency of which must be to mislead a jury on an important question, is discreditable to medical science. I have elsewhere pointed out the danger of relying upon the necessity of discovering what is called a fatal dose of poison in a dead body (ante, p. 184); but in this instance the line of defence was unjustifiable, since the quantity found in the body was sufficient to destroy life.

In reference to the fatal dose, the question may present itself, whether a diseased condition of the body may not, in some cases, augment the effects of the poison, and render a medicinal dose fatal. In the case of the late Dr. Chambers (April, 1853), Mr. Wilson found great disease of the heart. There was a strong odor of prussic acid on opening the body, and a phial which had contained prussic acid was found near. From the prescription, it appeared that the draught taken by the deceased had contained *six drops* of prussic acid, of Scheele's strength, ten drops of Battley's sedative solution of opium, some tincture of colchicum, and an ounce and a half of water. Mr. Wilson believed that deceased had taken this draught as a medicine, and that, owing to the latent disease of the heart, the prussic acid had caused death. A verdict was returned accordingly. In September, 1858, a similar inquiry was raised in the case of another medical gentleman,—Mr. Bainbridge,—who was found dead in his room, about three hours after he had been seen in his usual health. There was an ounce bottle of prussic acid, nearly full, on a table in the room, with a minim measure, a tumbler, and a decanter of water. He had been in the habit of taking prussic acid, and had that day written a prescription for himself, and had sent for the acid to a druggist's. On an inspection of the body, three or four days after death, there was found disease of the heart sufficient to account for death, and only traces of prussic acid in the stomach. It was suggested at the inquest that deceased had committed suicide, although there was an entire absence of motive. The strength of the acid in the bottle was lower than that which was stated to have been sent out by the druggist. In order to account for this it was surmised that deceased had poured off half the acid, and filled the bottle with water, thus lowering the strength. This hypothesis was considered to be supported by the fact that lime (from hard water) was contained in the prussic acid of the bottle; but, on examining the acid at the druggist's, lime was also contained in it. It was thought that there was a difference in the amount; but to have rested a presumption of suicide on such a minute difference as this would not have been justifiable. There was no reason to suppose that deceased had measured off half an ounce of the acid merely to throw it away, and had he swallowed it, more than traces would have been found in his body. The discovery of any quantity of this poison in a dead body is consistent with murder; but the discovery of mere traces, within a few days of death, is rather presumptive of an accident than of suicide; and when there is coexisting disease of the heart, we may fairly make allowance for a more energetic operation of the poison. The jury returned a verdict in accordance with this view. If, in the cases recorded by Mr. Garson, of Stromness (p. 571), and Dr. Geoghegan (p. 583), the persons had died instead of recovering from full medicinal doses, it would have been very easy to suggest that they had destroyed themselves; that each had taken a large dose, and that traces found in the bodies, two or three days after death, might be explained by the great volatility of the poison, and its loss by evaporation. It is obvious that in those two cases such a series of speculations would have been quite erroneous.

In reference to the medicinal use of this poison, there appears to be no strict relation between grains, minims, and drops. I have found by experiment, that sixty minims (or one drachm) of the same prussic acid, measured in three different measures, weighed respectively 61, 62·5, and 64 grains, while this quantity of the acid containing two per cent., dropped from an eight-ounce bottle, was equivalent to forty-two drops,—and the same measure of an acid at four per cent. was equivalent to forty drops. The volume and weight of drops vary according to the nature of the liquid, the size of the bottle, the width of the lip of the bottle, and the angle of inclination. Hence it is a most uncertain mode of measuring, and in reference to doses I have here substituted "grains" for "drops."

PERIOD AT WHICH DEATH TAKES PLACE.—It has been observed in the human subject, that when individuals have taken the same dose, death has occurred at very different periods of time: age, idiosyncrasy, the state of health, and the presence or absence of food in the stomach, are conditions which of course exert an appreciable influence on the operation of this poison. In the cases of the seven Parisian epileptics (ante, p. 566), a *similar* dose was given to all, but death took place at very *different* periods,—the first person died in about fifteen or twenty minutes, and the last only after three-quarters of an hour! In one instance in which seven drachms of the acid were taken, death took place in five minutes: in another, in which an ounce was taken, the person survived about ten minutes. (Sobernheim.) When the dose is two drachms and upwards, we may probably take the average period for death at from two to ten minutes. In Mr. Hicks's case, forty-nine drops of P. L. acid destroyed life in twenty minutes. It is commonly when the dose is in about a fatal proportion, that we find a person survives from half an hour to an hour. In this respect, death by prussic acid is like death by lightning: the person in general either dies speedily or recovers altogether. According to Dr. Lonsdale, death has occurred in the human subject as early as the *second*, and as late as the forty-fifth minute. There are cases recorded in which death has taken place after an hour, or longer (case of Marcooley, p. 565). The quantity here taken was probably two drachms (=2¼ grains of anhydrous acid). In the case of Montgomery (Glasgow Court of Justiciary, Dec. 1857), a similar dose was administered, and the evidence rendered it probable that the woman survived fifty minutes. In a case which occurred to Casper, a woman who had taken a large dose of the poison mixed with ethereal oil, was found lying dead, with a liquor-ladle in one hand, and half a cucumber in the other. (Ger. Med. i, p. 434.) The deceased was probably intoxicated when she took the poison; but the condition of the body showed a very rapid death.

Although it may be said that those who survive an hour have a good chance of recovery, yet death may occur from the poison after the lapse of several hours. In a case of poisoning by two ounces of laurel water, mentioned by Casper, a man, æt. 60, did not die until after the lapse of five hours, although he was rendered almost immediately insensible by the dose. (Ger. Med. 1857, p. 431.) Böcker met with a case in which an ounce of the acid of the Prussian Pharmacopœia (1 per cent.), equal to nearly *four grains and a half* of anhydrous acid, caused death after thirty-six hours. (Op. cit. p. 23.) Unless the greater part of the poison was vomited early, it is difficult to understand how it happened that the person could have survived so large a dose for so long a period,—one-fifth of this quantity having destroyed the life of an adult in twenty minutes (Mr. Hicks's case, p. 583). Böcker, however, appears to attribute death to the consequences of bleeding and the *nimia diligentia medicinæ!* But although death may not commonly ensue until after the lapse of some minutes or hours, insensibility, and consequently a want of power to perform acts of volition and locomotion, may sometimes come on in a few seconds. The time at which this loss of power is supposed to take place, has frequently become an important medico-legal question; and on the answer to it the hypothesis of suicide or murder in a particular case may rest. (See p. 568.)

TREATMENT.—Cold affusion to the head and spine has been found the most efficacious mode of treatment. In a case that occurred to Dr. Banks, a girl took by mistake in medicine thirty minims of prussic acid. Immediately afterwards she sprang up convulsively from her seat, and fell senseless. Her teeth were firmly set, and her eyes staring and fixed. Stimulants failed to rouse her: the limbs were flaccid; the pupils dilated, and she was wholly insensible; the respiration was slow, and the pulse scarcely perceptible. A stream of cold water from a pitcher was allowed to fall from some height on the region of the spine. In a minute she began to move, and became convulsed; her symptoms abated, and in a few hours she was quite collected. She recovered in a few days; but there is hardly a doubt that she would have died, had she not been thus treated. (Ed. Med. and Sur. Jour. xlviii, 44.) In the following case of recovery, cold affusion was resorted to at a later period. (Med. Gaz. xxxvi, 104.) H. G., æt. 59, a healthy laborer, swallowed by mistake a dose of anhydrous prussic acid, equal to nine-tenths of a grain, in eighteen drachms of distilled water. He had no sooner taken it than he was seized with a violent constriction of the diaphragm, and a sense of suffocation. He walked to the outer door of the house, about twelve or fourteen paces, when he fell insensible, and, in his fall, broke a large pan which was full of water, the contents of which saturated his clothes; this, no doubt, had a beneficial influence upon him. The accident occurred about half-past seven o'clock in the morning, and it was not until twenty minutes after eleven, or nearly four hours after taking the acid, that he showed symptoms of returning animation; when, by the application of cold water and ammonia, he was soon restored. On coming to himself, he vomited freely; but no odor of the acid was perceptible in the liquid thrown from the stomach. The next day he had recovered. Mr. Harthill has reported a case in which cold affusion led to recovery after probably a large dose. (Prov. Med. Jour. March 5, 1845, p. 153.)

In Mr. Garson's case (Ed. Med. and Sur. Jour. lix, 72, and p. 571, ante), cold affusion and the use of ammonia appear to have been attended with the best effects. The vapor of ammonia may be cautiously applied to the nostrils, and stimulating liniments by friction to the chest and abdomen; but unless the dose is small, and the patient is seen early, there can be little hope of benefit from any treatment. Internal remedies appear to be of no service. The blood is speedily poisoned, and no chemical antidote can reach this liquid to counteract the effects of the poison. If the power of swallowing remains, an emetic may be given, the stomach-pump used, or the throat irritated, to clear the stomach of any residuary poison. Turpentine, or other stimulating injections, may be administered if the state of the patient admits of such treatment. The mixed oxides of iron (prepared by adding carbonate of soda to a strong solution of green vitriol and persulphate of iron), have been proposed as an antidote. They can be productive of no injury, and may, to a certain extent, fix the unabsorbed prussic acid in the stomach; but the patient is not commonly seen until it is too late to administer this or any other compound. The power of swallowing is lost.

Bleeding from the jugular vein was strongly recommended by Dr. Lonsdale. It may be beneficial in the after-treatment of protracted cases when there is any sign of cerebral congestion; but, in general, the pulse is scarcely perceptible, and bleeding is therefore inapplicable. There is always great loss of power under the operation of this poison: hence, the imprudent or hasty abstraction of blood may prevent recovery. Experiments on dogs show that this mode of treatment is not attended with any benefit. (Prov. Trans. N. S. iii, p. 72.) In one of the Parisian cases (p. 566), death took place soon after a small quantity of blood was drawn from the arm; and, according to Böcker, bleeding from the arm accelerated death in a protracted case of poisoning observed by him. In his opinion, it is a decidedly injurious practice. (Op. cit. 127.) Viewing the whole of the blood as a poisoned fluid, it does not seem reasonable to resort to bleeding in the early stage; and in the later stage, it may either retard recovery or accelerate death.

In Reg. v. Belaney (Cent. Crim. Court, Aug. 1844), a question arose respecting the proper mode of treating cases of poisoning by prussic acid. The prisoner was a surgeon, and he was charged with the murder of his wife, who died in his presence from the effects of a large dose of prussic acid. The medical facts in the case were very simple. There could be no doubt that the poison had been taken, and that it was the cause of death. The nature of the symptoms, their rapid and fatal course, and the detection of the poison in large quantity in the stomach, rendered these conclusions absolutely certain. Again, there was no doubt that the poison had been administered, either intentionally or unintentionally, by the prisoner, *i. e.* that it was through his act, either criminal or innocent, that the poison was placed within reach of the deceased, and under circumstances which would render it not improbable that she would swallow it by mistake. It was placed in a common drinking-glass in the bedroom,—the prisoner being at the time in an adjoining room. He accounted for this strange conduct, by saying that he was in the habit of using prussic acid medicinally,—that he broke the bottle in trying to remove the stopper; and, in order to save the contents, collected the acid in a tumbler or glass, such as is used for drinking water! His attention was called off, and he went into an adjoining room, without, as it would appear, making any remark, or cautioning his wife respecting the poison placed in the tumbler, and within her reach!

The presumption of criminality, under such circumstances, had no direct relation to medical evidence: it was a question to be decided by the jury from the facts proved. The medical evidence had, however, two important bearings: 1, the plan of treatment which should be adopted in such an emergency by a medical man; 2, the exact period at which insensibility and loss of consciousness supervene in cases of poisoning by prussic acid. The prisoner, on finding that his wife had swallowed the poison, called for assistance, but did not at the time state the real cause of the symptoms; although it came out in evidence that he must have known that the deceased had swallowed prussic acid. He caused her feet and hands to be put in hot water, and talked of bleeding her; but said it was of no use, as circulation had ceased ("she had no pulse"). He told the first witness who came to her, that "she would not come to,—it was a disease of the heart, and that her mother had died just like it nine months ago." But it was subsequently proved that the prisoner had himself registered the cause of death in the mother, as bilious fever! The late Dr. A. T. Thomson, who gave evidence at the trial, was questioned upon the usual remedies in such cases, which he stated to be,—cold affusion, ammonia, and stimulants; and very properly expressed an opinion, that what had been done by the prisoner could be of no benefit whatever. The jury acquitted the prisoner. The verdict did not proceed from any defect in the medical evidence; the cause of death was clear, but it was for the jury to determine the value of the moral and circumstantial evidence against the prisoner as the alleged administrator. Of these circumstances, which were exceedingly strong, it is here unnecessary to speak; but the learned judge and the jury, in the opinion of most persons, took a very lenient view of them.

CHAPTER XXXVII.

PRUSSIC ACID—CHEMICAL ANALYSIS—THE SILVER, IRON, AND SULPHUR TESTS APPLIED TO THE LIQUID AND VAPOR—OBJECTION—DETECTION IN ORGANIC LIQUIDS WITHOUT DISTILLATION—PROCESS BY DISTILLATION—DETECTION IN THE TISSUES—PERIOD DURING WHICH IT MAY BE DETECTED IN THE DEAD BODY—ALLEGED SPONTANEOUS PRODUCTION OF PRUSSIC ACID—THE POISON NOT DETECTED—QUANTITATIVE ANALYSIS.

CHEMICAL ANALYSIS.

PRUSSIC ACID is a limpid, colorless liquid. Its specific gravity, when its strength ranges from 2 to 5 per cent. of anhydrous acid, is 0·998. It is, therefore, just barely lighter than water, but it readily mixes with water and alcohol in all proportions. It has a faint acid reaction; if litmus paper is strongly reddened by it, the presence of sulphuric or of some foreign acid may be suspected. It is sometimes thus acidulated for the purpose of preserving it. Its vapor has a peculiar odor, which, when the acid is concentrated, although not at first perceptible, is sufficient to produce giddiness, insensibility, and other alarming symptoms. (See ante, p. 563.) It was at one time supposed that the *odor* might be present in cases in which it would be impossible to detect the poison by chemical processes, and instances of this are given by Orfila (Ann. d'Hyg. i, 489), by Dr. Lonsdale (Ed. Med. and Surg. Jour. li, 52), and by Dr. Christison (Op. cit. 1854, pp. 760, 774). Improved methods of research have, however, shown that the acid may be detected in cases in which no odor is perceptible.

The tests which are best adapted for the detection of this poison, either in liquid or vapor, are equally applicable, whether the acid be concentrated or diluted, and, so far as the detection of the *vapor* is concerned, whether it be pure or mixed with organic matter. *In the simple state*, the tests are three in number: the *Silver*, the *Iron*, and the *Sulphur* tests.

1. *The Silver test.—Nitrate of Silver.*—This yields, with prussic acid, a dense white precipitate, speedily subsiding in heavy clots to the bottom of the vessel, and leaving the liquid almost clear. The precipitate is identified as cyanide of silver by the following properties: *a.* It is insoluble in cold nitric acid; but when drained of water, and a sufficient quantity of strong acid is added, it is easily dissolved on boiling. *b.* It evolves prussic acid when digested in muriatic acid. *c.* The recent precipitate is readily dissolved by a few drops of strong solution of potash, provided the whole of the prussic acid has not been precipitated by nitrate of silver. If to this mixture a small quantity of a solution of green sulphate of iron is added, and the whole well shaken, Prussian blue is formed, and is made evident on adding diluted sulphuric or hydrochloric acid to the turbid mixture. The acid dissolves the surplus oxide of iron, and leaves the Prussian blue in the precipitate. *d.* The precipitated cyanide of silver, when *well dried* and heated in a small reduction tube, yields cyanogen gas, which may be burnt at the mouth with a rose-red flame and blue halo. This is a well-marked character, and at once identifies the acid which yielded the precipitate as prussic acid. By this property the cyanide is eminently distinguished from all the other salts of silver. The tenth of a grain of cyanide of silver is sufficient for the performance of this experiment. This represents 1-50th of a grain of prussic acid or one drop of pharmacopœial acid.

In the employment of the silver test for the detection of the *vapor* of the poison, we may place a few drops of the silver solution in a watch-glass, and invert this over another watch-glass containing the suspected poisonous liquid. Cyanide of silver, indicated by the formation of an opaque white film in the solution, is immediately produced, if the acid is only in a moderate state of concentration. One drop of the pharmacopœial acid (equivalent to 1-50th of a grain of pure acid) produces speedily a visible effect. When the prussic acid is much diluted, a few minutes are required; and the opaque film begins to show itself at the edges of the silver solution. In this case the action may be accelerated by the heat of the hand. If the vapor be allowed to act very slowly and in a diluted form, on the nitrate of silver, the cyanide is produced in well-defined prismatic crystals, plainly visible under a low power of the microscope. This is an additional special character of this compound. Further, unlike the chloride of silver, the cyanide remains white under exposure to light, while the chloride rapidly acquires a slate-gray color.

2. *The Iron test.*—The object of the application of this test is the production of *Prussian Blue.* We may add to a small quantity of the suspected poisonous liquid a few drops of potash, and of a solution of green sulphate of iron. A dirty green or brownish precipitate falls; on shaking this for a few minutes, and then adding diluted muriatic or sulphuric acid, the liquid becomes blue; and Prussian blue, of its well-known color, unaffected by diluted acids, slowly subsides. If the prussic acid be in small quantity, the liquid has at first a yellow color, from the salt of iron formed; it then becomes green, but the precipitate ultimately subsides so as to appear of a blue color in the mass. The same result is obtained by adding a solution of the iron-salt to the potash-solution of the cyanide of silver (supra); and thus, by this method, the two tests may be applied to only *one* portion of the poison. The iron test may be employed for the detection of the *vapor* of prussic acid, by the process described in speaking of the silver test. For this purpose we place a few drops of caustic potash in a small white saucer or watch-glass, and invert it over the suspected liquid. After a few minutes a drop of a solution of green sulphate of iron may be added to the potash, and then a drop of diluted muriatic acid,—Prussian blue will appear if prussic acid is present. The recently precipitated mixed oxides of iron with potash may be placed in the upper watch-glass with the same results. The silver and the iron tests may be easily conjoined in testing the same quantity of poison. If the precipitated cyanide of silver, obtained by the addition of nitrate of silver to the suspected liquid, be moistened with strong muriatic acid, and the vapor of prussic acid evolved be collected in a watch-glass or saucer, on the plan just described, Prussian blue will be procured, and the action of the silver test will be strongly corroborated. The iron test will detect as small a quantity of the acid as the silver test.

3. *The Sulphur test.*—Liebig first proposed the following process for detecting prussic acid as a *liquid.* (Oesterreichische, Med. Wochenschrift, 27 Marz, 1847, 396.) If a few drops of bihydrosulphate of ammonia (containing a little excess of sulphur) be added to a small quantity of the solution of prussic acid, and the mixture be gently warmed, it becomes colorless, and, on evaporation, leaves sulphocyanate of ammonia—the sulphocyanic acid being indicated by the intense blood-red color produced on adding to the dry crystalline residue a solution of colorless persulphate of iron: the color immediately disappears on adding one or two drops of a solution of corrosive sublimate. The intensity of the color is much lowered by moderate dilution with water. This process is very delicate, and it therefore requires some care in its application: thus, if too large a proportion of the hydrosulphate of ammonia is added,—or if the boiling and evaporation be not carried far enough,

the persalt of iron will be precipitated black by the undecomposed hydrosulphate of ammonia; and, if the heat be carried too far, the sulphocyanate of ammonia may itself be volatilized and lost. This test requires a longer time for its application than either the silver or the iron test. Should the prussic acid contain traces of Prussian blue or a salt of iron, it may acquire a dark color on the addition of hydrosulphate of ammonia.

Liebig confined the application of this test to liquid prussic acid, but I have found that it is applicable to the detection of the minutest portion of the *vapor*. In this respect it is as delicate for prussic acid as Marsh's process for arsenic. It will reveal the presence of the 4000th part of a grain under favorable circumstances. In order to apply it, we place the diluted prussic acid in a watch-glass, and invert over it another watch-glass, holding in its centre one drop of the bihydrosulphate of ammonia. No change apparently takes place in the hydrosulphate; but if the watch-glass be removed after the lapse of from half a minute to ten minutes, according to the quantity and strength of prussic acid present, and gently heated to 100°, sulphocyanate of ammonia, sometimes in a crystalline state, will be obtained on evaporating the drop of hydrosulphate to dryness. With an acid of from three to five per cent. the action is completed in ten seconds. The addition of one drop of persulphate of iron to the dried residue brings out the blood-red color instantly, which is intense in proportion to the quantity of sulphocyanate present. Such is the simple method of employing the test. When the prussic acid is excessively diluted, the warmth of the hand may serve to expedite the evolution of the vapor, but a moderate heat (80° to 100°) is afterwards required to effect the change. (See Med. Gaz. vol. xxxix, p. 765.)

In detecting the *vapor*, the sulphur test acts, *cæteris paribus*, more rapidly and more delicately than the silver test; but the two may be usefully employed together. Add to the deposited cyanide of silver one drop of hydrosulphate of ammonia and warm the mixture. Persulphate of iron will produce a red color in the liquid in spite of the presence of black sulphuret of silver. The silver test acts *visibly*, and therefore serves as a guide; the sulphur test acts invisibly; for there is no apparent change unless the glass be left so long that the ammonia is spontaneously evaporated, and the sulphur oxidated or deposited. The sulphur process will enable an analyst to detect the vapor of prussic acid when no odor can be perceived, and when the silver and iron tests fail to act with certainty. After the exposed mixture has been heated, the acid is solidified, and the quantity for testing thus concentrated into the smallest possible bulk. In reference to putrescent liquids, it is not affected by sulphuretted hydrogen like the silver and iron tests, and it acts in spite of the presence of this gas.

Objections.—Nitrate of silver, it is well known, gives a white precipitate with numerous acids; but if the properties of the precipitate, especially those marked under *b*, *d* (p. 590), be observed, there is at once an end to any objection; a volatile acid containing *cyanogen* must have been present in the liquid. The production of Prussian blue from the liquid, in the manner described (p. 591), is free from any chemical objection. There is no liquid acid known to the chemist that will, under similar circumstances, produce the same results. The sulphur test is also free from any objection,—the sulphocyanate of iron being well characterized by its blood-red color, its solubility in a solution of corrosive sublimate, and by other chemical properties.

When the tests are applied to the detection of the *vapor*, they are absolutely free from any chemical objection. There is no other acid, or volatile compound of cyanogen, which in any respect resembles prussic acid, that is known to produce similar results. The tests applied to the *vapor* are therefore *conclusive*. There can be no pretence for omitting this part of the process in any medico-legal analysis.

In organic liquids.—Any organic liquid suspected to contain prussic acid, *e. g.* the matters first vomited, or the contents of the stomach after death, may, under the limitations already mentioned (p. 567), have an odor of the poison perceptible to one or more individuals. If the liquid has no odor of prussic acid, but an odor of sulphuretted hydrogen, or of some strongly smelling substance, *e. g.* peppermint or tobacco, still the poison may be present, and it may be detected, if not as a liquid, by the ordinary process of distillation—at least by its *vapor*. Of the two processes to be pursued, that which relates to the detection of the *vapor* is the more certain, and open to the fewest objections. It should always be tried before resorting to distillation, because no plausible objection can then be raised on the ground that prussic acid might have been generated from a decomposition of animal matter during the process. If the poison be clearly and unequivocally detected by its vapor, there is no necessity for resorting to distillation, except for the purpose of determining the proportion of prussic acid present.

Detection by vapor without distillation.—The organic liquid may be placed in a wide-mouthed bottle, to which a watch-glass has been previously fitted as a cover. The capacity of the bottle may be such as to allow the surface of the liquid to be within one or two inches of the concave surface of the watch-glass. The solution of nitrate of silver is then used as a trial-test in the manner already described (page 590). If the 1-200th of a grain of prussic acid be present and not too largely diluted, it will be detected (at a temperature of 60°) by the drop of nitrate of silver being converted into an opaque white film of cyanide of silver, the chemical change commencing at the margin. This, if in small quantity, may be found to be soluble in concentrated nitric acid: the solution is aided by heat. Owing to the evolution of sulphuretted hydrogen, the nitrate of silver may be darkened. We must then at once employ the sulphur test, as this is not affected by sulphuretted hydrogen gas. Any black precipitate formed by the addition of a persalt of iron may be removed by muriatic acid.

Detection by distillation.—The organic liquid may be distilled without any mixture of acids or alcohol, in a water-bath, at 212°, and from one-sixth to one-eighth of the contents of the retort collected through a condensing tube in a receiver kept cool by water. The test may be applied to the distilled liquid. If the trial-test indicate that the quantity of poison is small, a few drops of a solution of nitrate of silver or of caustic potash may be placed in the receiver, to fix the acid as it is distilled over; Prussian blue may then be procured by the process described, or the vapor may be at once absorbed by hydrosulphate of ammonia in the receiver, and the liquid evaporated at 100° to obtain sulphocyanate. This is rarely required, as the liquid may be easily obtained by the first, or by a second distillation, in a state fit for the application of the tests.

Detection of prussic acid in the tissues.—That prussic acid is absorbed, and widely diffused through the body is proved by experiments elsewhere related (page 72). The acid is also rapidly eliminated. Prussic acid has been detected in the blood and viscera of animals poisoned with it, by the process of distillation. Kramer thus discovered it in the blood of an animal. Heller detected the acid in most of the soft organs, as well as in the blood, by digesting them in a small quantity of potash, and then adding a salt of iron and muriatic acid. (Archiv. für Phys. Chem. 1845, i, and ii, 143.) Mr. Hicks states that he poisoned a cat with twenty minims of Scheele's acid. Eight hours after death the body was examined; a small aperture was made in the chest, and a watch-glass, containing a few drops of nitrate of silver, was accurately fitted over it. In half an hour there was a perceptible white margin, and in an hour the solution of the nitrate had been converted to a white film, proved to be cyanide of silver by its insolubility in cold, and its solubility in boiling nitric acid. The whole of the contents of the chest, when placed in a bottle, gave out a vapor which readily produced with the respective tests cyanide of silver and Prussian blue. (Med. Gaz. xxxvi, 632.)

In addition to the facts elsewhere related, regarding the absorption and diffusion of prussic acid, the following experiments have a special interest, as they demonstrate the rapidity of this diffusion. Dr. C. Willis, of Dublin, injected one drachm of diluted prussic acid (4 per cent.) into the pleura of a rabbit. Symptoms of poisoning began in ten seconds, and the animal died in twenty-five seconds from this time. When the symptoms commenced, a string, previously placed around one of the hind legs, was drawn so tightly as to stop all circulation. The leg was immediately cut off below the ligature, and laid by in a little water well covered up. The next day, although no odor of prussic acid could be detected, it was readily found by distillation in a water-bath and the application of the iron test, as well as by the sulphur test. In another experiment the symptoms began and the leg was tied in *eight* seconds. Death ensued in thirty seconds. Prussic acid was detected in the amputated leg as well by the odor as by the tests. In a third experiment on a puppy, five days old, symptoms began and the leg was tied *four seconds* after the injection; the presence of the acid was demonstrated both by the sense of smell and tests. (Lancet, May 29, 1858, p. 541.)

The poison may be detected in the blood, secretions, or any of the soft organs, by placing them, if recent, in a bottle, and collecting the vapor in the manner already described (p. 591). This will be found to be more convenient and satisfactory than the process of distillation. In the case of a dog poisoned by prussic acid, Mr. Hicks brought me the stomach after it had been exposed twenty-four hours and thoroughly washed under a current of water, and yet the poison was readily detected by placing the whole organ in a bottle, and absorbing the vapor by nitrate of silver. This shows how completely the animal tissues are penetrated by prussic acid, and how, at least for a time, it is firmly retained by them. Its great volatility, however, is also proved by the detection of the vapor under these circumstances.

It is important to consider, in reference to the detection of this poison in organic liquids or solids, on what results an analyst should rely for conclusive evidence of its presence. In the application of the tests for the vapor, either to an organic liquid or to the product of a double distillation, it appears to me that in order to render a medical opinion conclusive and satisfactory, results should at least be obtained by the silver and sulphur,—or iron and sulphur tests. So small a quantity of the poison is required for the application of two tests, that there seems to be no good reason for relying upon the action of one. The silver and iron test may be applied first, and these should be followed by the sulphur test, as the latter always contaminates the liquid to be tested. It is true that the sulphur test will reveal the presence of the poison when the iron test fails, owing to the difficulty of collecting a minute trace of Prussian blue: but in this case the quantity of poison must be exceedingly small, and the reaction of the sulphur test very feeble. Although we at present know of no vapor but that of prussic acid which will thus affect the sulphur test, it appears to me that we should not be justified in relying upon infinitesimal results, which admit of no kind of corroboration. The question is here much the same as in reference to the detection by the process of Marsh, of minute traces of what is alleged to be arsenic, when the quantity is too small to be separated by Reinsch's process. The silver test cannot be relied on for detecting small quantities of prussic acid in organic liquids or solids, unless the film of cyanide of silver is converted to sulphocyanate of ammonia by the method described (p. 592). When, however, we have procured the colored results by the iron and sulphur tests, there can, it appears to me, be no reasonable doubt of the presence of the poison. With either result separately, as applied to the vapor, there may be room for objecting to the conclusion, that prussic acid has been certainly detected. If more than one test is used by a cautious analyst in examining the liquid poison when in appreciable quantity, how much more is such a corroboration required when he is dealing with an imponderable quantity of vapor obtained from the organic liquids or solids of a dead body?

Period at which the acid may be detected after death.—If an organic liquid containing the poison is kept in a closely stoppered vessel it may sometimes be detected after a long period. When the liquid has been exposed for three or four days the vapor tests have ceased to indicate its presence at the mouth of the bottle containing the liquid: but the acid has been obtained by distillation. I have detected prussic acid when mixed in small quantity with porter after the lapse of twelve months; but then the bottle had been kept closed, and the contents were not putrefied. The practical question for consideration is, for how long a period may we expect to find the poison in the contents of the putrefied stomach or tissues of a dead body. The following facts throw some light upon this question: The acid has been found in the stomach by distillation *seven* days after death, although the odor could not be perceived before distillation. (Case of Ramus, p. 580.) Orfila is said to have discovered it *eight* days after death in the cases of the Parisian epileptics; but he merely states he perceived an odor of bitter almonds, not that he obtained the poison by distillation! (See p. 577.) In a case in which three drachms had been taken, I could neither detect the acid by the odor nor by the most careful distillation, *twelve* days after death. (Guy's H. R. April, 1845.) The poison has been detected in the stomach by the vapor and by distillation in one case in three, and in other cases in four, five, and ten days after death. (See Essential Oil of Almonds, p. 618.) In the case of Marcooley (Cent. Crim. Court, Dec. 1856) the contents of the stomach consisted of two ounces of a brownish fluid mixed with oil. They were received by the chemical witness, *seven days* after death, in a bottle secured with bladder only, at the mouth. Still he detected prussic acid faintly by the smell but distinctly by the tests. He procured from the stomach and contents a quantity of prussic acid equal to six-tenths of a minim of Scheele's strength. In this instance about two drachms of the poison had been taken, and had caused death in an hour. In the case of Montgomery (Report of Trial of Thompson, Glasgow Circuit Court, 1857, by Hugh Cowan, pp. 9 and 53), the deceased died in about fifty minutes after having taken two drachms of prussic acid (three and a quarter grains of anhydrous acid). The death took place on the 13th September: the body was buried on the 17th, and exhumed on the 30th. The parts removed were then put into stoppered bottles, and on the 5th of October the Drs. McKinlay detected prussic acid doubtfully by the odor, but distinctly by the three tests, in the stomach, before distillation, as well as in the liquid distilled from the stomach and its contents. They did not succeed in detecting its presence in the tissues. About five weeks subsequently to this analysis, the organs, which had been kept closely secured in glass bottles, were examined by Dr. Maclagan. The heart, kidneys, and intestines gave no indication of the presence of the poison, but it was detected by the sulphur test, in the form of vapor, in one half of the spleen (p. 62) although there was no odor of the poison. Dr. Maclagan quoted in his evidence a French case, on the authority of a M. Brame, in which prussic acid is said to have been detected in a dead body *twenty-one days* after interment. (Wharton and Stillé, Med. Jur. p. 492.) But the steps of the analysis in this case are so unsatisfactory as to render it doubtful whether prussic acid was discovered at all. A mixture of chloride of silver and animal matter would have given rise to the reactions obtained by M. Brame. In the former edition of this work, as well as in the Medical Jurisprudence, I have quoted, on the authority of Mr. Nunneley, a case in which the late Mr. West is reported to have detected prussic acid by distillation in the stomach of a man twenty-three days after death, "although no pains had been taken to preserve it." (Prov. Trans. 1847, N. S. vol. iii, p. 81.) Mr. Nunneley ascribes the preservation of the acid in the contents of the stomach to their being strongly imbued with gastric juice, &c. The facts, however, when closely examined, do not support either the statement or the theory. The contents of the stomach, examined twenty-four hours after death, smelt strongly of prussic acid, and yielded clear evidence of the presence of the poison by all the tests applied to the vapor. The whole quantity of anhydrous prussic acid in the stomach, as determined by distillation, was calculated to amount at this time to only four-tenths of a grain. It appears that Mr. West distilled another portion of the contents twenty-three days after death, and the tests then acted upon the distillate as readily as at first. (Prov. Med. and Surg. Jour. July 23, 1845, p. 466.) It is not stated that no care had been taken to preserve them; and it is most improbable that if they had been exposed, any part of the four-tenths of a grain of this volatile poison would have remained in them after the lapse of three weeks. This case merely proves, therefore, that prussic acid may be retained and detected after many days in the contents of the stomach of a person who has taken this poison. The longest period at which it has been clearly detected in the dead body was in the case of Montgomery (supra), namely, *seventeen days.* It appeared from the evidence in this case, that the body was placed under favorable circumstances for the retention of the poison.

It is obvious that the period at which this poison may be detected, must depend on the dose taken, the duration of life after it was taken, and the circumstances under which the body has been placed after death. In the case of Walter Palmer (Rugeley inquest, January, 1856, p. 182), a question arose whether, assuming that the deceased had taken just a fatal dose of Scheele's prussic acid, *i. e. twenty minims*, or about one-third of a teaspoonful, it was probable any part of the dose would remain in the stomach *four and a half months* after death. Although deceased was in a diseased condition, there was a strong probability from moral circumstances that he had died from the administration of a dose of prussic acid in brandy. Dr. Rees and I examined the stomach by the vapor tests, as well as by distillation, soon after the body was exhumed: but there was no trace of prussic acid or of any sulphocyanide in the stomach. The body had been buried eighteen weeks, and was far advanced in putrefaction. There was reason to believe, on the theory of poisoning, that the dose given was small. It was, therefore, not in the least degree probable that any trace of prussic acid or of the results of its decomposition would be found. Mr. Herapath, of Bristol, however, circulated a statement that he had detected prussic acid in a dead body at a long period after death, and led a section of the public to believe, that because it was not found in the body of Walter Palmer, there had been no poisoning! On examining the grounds on which this statement was made, they wholly fail to support the singular view adopted by this chemist. It does not appear that he has ever detected prussic acid in a dead body, even one month after death: but in one case of poisoning by the oil of bitter almonds, having failed to detect the poison in the stomach, he inferred that the oil had been taken, in consequence of his having found sulphocyanic acid in the *blood* two months after death (ante, p. 182; Chemist, 1854, vol. i, p. 321). Assuming the correctness of this conclusion, and that the witness was justified in inferring poisoning by oil of bitter almonds through the stomach from this alleged chemical discovery in the blood, it by no means follows that a minimum fatal dose of prussic acid should leave any trace of its presence in a stomach or in the blood nearly *five months* after death!

Is the discovery of prussic acid in the stomach or tissues of a person, a proof that death has been caused by it? As a general rule, we should be justified in answering this question in the affirmative; as less than a grain may destroy life. We do not here meet with the objection which applies to most other poisons, that the patient may have been cut off by disease supervening after it had been taken; since if the poison operates fatally at all, it is in the course of a few minutes. Latent diseases of the heart and brain may undoubtedly by a coincidence cut short life. Prussic acid, it is well known, is used medicinally, and patients are in the habit of taking it for some time after they have ceased to be attended by the medical man who has prescribed it. A person may die suddenly from natural disease while taking the acid, and a chemist, relying on the discovery of the poison in the stomach, might give an opinion that death was caused by it. In such cases, on recent inspection, the acid will be found in mere traces: if a larger quantity is detected than would correspond to a medicinal dose, there may be reason to suspect death from accident or suicide: but the whole of the facts of the case should be considered, or the results of a chemical analysis might seriously mislead a jury.

The detection of the acid in the tissues, in the soft organs, or in the blood, would justify the opinion that it had been taken into the body during life. It is a diffusible poison, and will readily traverse the membranous structures; but it must be within the body in order that this should occur, and the only other supposition of which the facts will admit is, that the acid was injected into the stomach after death. When two or more poisons are present in the stomach, and one is prussic acid in a fatal dose, there can be no reason to hesitate in assigning death to the latter. In a case which occurred in 1837–8, prussic acid and arsenic were found in the stomach after death. In another, the mixture taken by the deceased consisted of brandy, opium, arsenic, and prussic acid.

Alleged spontaneous production of prussic acid.—If an analyst confined his processes to the detection of free prussic acid in the stomach, it might be objected to his results that it had been spontaneously generated there. If he detected it only *after* distillation, it might be alleged that the heat employed had reacted on the organic matter and produced it. Supposing that the presence of the poison has been previously determined in the blood and tissues by the *vapor* tests, it is obvious that this objection would at once fall to the ground. This part of the evidence may be neglected, or it may fail: hence it is necessary to consider whether prussic acid can be spontaneously generated, under any circumstances, in organic liquids or in the body, within a few days after death.

That this acid is a product in various chemical processes is undoubted; thus it appears to be formed at a high temperature by the reaction of nitric acid on alcohol (see p. 564), as well as in other cases. No one has yet suggested that there is *normal* prussic acid in the body; hence the whole question turns upon the chemical probability of its spontaneous generation from articles of food or other substances which may be contained in the stomach. That the acid should be thus produced without the aid of heat, has not, so far as I am aware, been suggested; and if it were liable to be produced by heat during distillation, it is obvious that those who are engaged in medico-legal researches would be continually meeting with it as a product. There is nothing to bear out such an hypothesis in works on chemistry or toxicology. If the organic liquid contained any of the kernels of certain fruits, the pulp of the black cherry, or laurel berries,—bitter almonds or substances which are known to furnish prussic acid by mere contact with water, the poison would be undoubtedly procured by distillation; but the result is then artificial, and cannot constitute a valid objection to the chemical processes employed. Orfila believed it possible that prussic acid, like acetic acid or ammonia, might be a product of decomposition in the dead body; or, at any rate, that putrefaction might engender some compound which would give with the different tests all the reactions of prussic acid. (Toxicologie, ii, p. 405.) This is put as a bare possibility from analogy; and as a corrective to the doubt thus thrown on chemical evidence, he says the objection is inadmissible when the person has died under symptoms of poisoning by prussic acid, and when the appearances in the body are like those caused by this poison! This, however, would not meet those cases where nothing was known concerning the symptoms. He further states, that if an organic liquid is not much decomposed, and only a gentle heat is applied during distillation, the objection cannot hold, because the poison is not obtained under these circumstances. Orfila does not, however, adduce a single fact in support of his views; and the result of the only experiment which he mentions is decidedly against them. M. Bonjean, of Chambery, has given the results of some experiments on the distillation of beef with water, and on the blood of the ox; also on various mixtures of organic matter, and of animal substances, in a state of putrefaction (op. cit. p. 54); but in not one instance was prussic acid found to exist, except when a substance containing it had been added! The results of experiments do not bear out this hypothesis; and if analogy is to be substituted for proof in its favor, there is nothing to prevent the admission of the production of morphia, strychnia, and other organic poisons as a result of putrefaction! The alleged production of prussic acid by the mere distillation of organic matter under a temperature of 212°, appears, therefore, to be an unfounded objection. During a period of twenty-seven years, I have distilled the contents of numerous stomachs, in all states of decomposition,—organic liquids, such as porter, milk, gruel, either mouldy or putrid, without in any one instance finding the slightest trace of prussic acid in the distilled liquids, either by the odor or by the usual tests; and it appears to me that until some unequivocal results in support of this hypothesis are forthcoming, a medical witness would be fully justified in utterly rejecting it as an unfounded assumption.

When the stomach contains matters from which prussic acid is known to be chemically producible by simple admixture with water, with or without the application of heat, the case is different. It then becomes a question whether the quantity of prussic acid which these substances are capable of yielding, bears any proportion to the actual quantity detected. One bitter almond will give a perceptible quantity of Prussian blue. Dr. Guérard states, on the authority of M. Filhol, that nearly all vegetable substances containing nitrogen possess the property of transforming *amygdaline* to essential oil of bitter almonds and prussic acid. A mixture of common flour, barley, or rye, produced this effect in five or six hours; but with maize, it required a period of three or four days. He then inquires whether a stomach might not possibly contain the materials which would lead to this transformation; and in the event of sudden death from natural causes, whether there might not be a false accusation of poisoning by prussic acid? (Ann. d'Hyg. Oct. 1847, p. 442.) If the human stomach contained any substance taken as *food* or medicine, from which prussic acid was producible, such a mistake might occur; but M. Filhol's experiments show that *amygdaline* must be present; and this peculiar body is not a constituent of ordinary food. It is found in the bitter almond, in peach kernels, in the berries and leaves of the laurel, and I have found it in the fruit of the passion flower. If these substances have not been eaten with the food, or not taken in sufficient quantity to account for death, no mis[illegible] can arise; if, however, they have been taken, then the case resolves itself into one of poisoning by these substances. If a person has swallowed pure amygdaline, the result would be the same; but the real force of this objection to chemical evidence is not to produce prussic acid from amygdaline, but to produce amygdaline from ordinary food, or by common putrefactive fermentation. It is as easy to conceive that prussic acid should be formed in the stomach at once from beef, potatoes, and [illegible]her substances, as that amygdaline should be derived from them. It may be proper to state that sweet almonds and common nuts contain no amygdaline; its presence in any seed is indicated by the powerful odor of the essential oil of almonds on trituration with *cold* water. (See Med. Gaz. vol. xxiii, p. 828.) Dr. Witling is represented to have found prussic acid as a spontaneous product of the decay of unsound cheese. (Christison on Poisons, p. 756.) I have examined cheese in various stages of decay, but not the slightest trace of prussic acid has been detected in it by the most delicate tests. Large quantities of decayed cheese are sold to the poor in London, but we never hear of any effects like those caused by prussic acid resulting from its use.

Notwithstanding the entire absence of proof that prussic acid is generated, either at common temperatures or at a heat of distillation (212°), it is necessary to state that a salt (sulphocyanide of potassium) containing cyanogen, exists in small quantity in the saliva. This may be mixed with the contents of the stomach, and it may be suggested that it would yield prussic acid by distillation with an acid. In answer to this it may be stated, that if the vapor tests have given the usual results before distillation, the objection cannot hold, because the sulphocyanic is a fixed acid in a state of combination. If it happened that any ferrocyanide or sulphocyanide of potassium was present in the stomach at the time of death, the distillation of the contents with sulphuric, phosphoric, or tartaric acid, as recommended by some analysts, would lead to the production of prussic acid. These salts, however, are not used in medicine: if present, their presence would be easily detected on neutralizing the undistilled liquid of the stomach (if alkaline) and adding persulphate of iron: this would at once produce Prussian blue or a deep red color. It is, however, unnecessary to employ an acid in distillation except to neutralize the liquid if alkaline. No advantage is gained by the addition of alcohol or any foreign substance to the contents of a stomach under these circumstances. They should be dealt with in the state in which they are removed from the body. Cyanide of potassium may be present in the stomach and yield prussic acid by distillation: but this is an active poison, and its presence may be easily discovered in the liquid contents before they are distilled. In all cases in which the vapor tests fail to act, it is advisable to test the liquid before distillation, for the presence of a cyanide or a sulpho- or ferrocyanide.

This question has more than a theoretical interest. In two important cases of murder in recent times, the alleged spontaneous production of prussic acid in a dead body was made a ground of defence, although unsuccessfully. As a last resort in a desperate defence, a medical witness must be prepared to hear this objection urged against his chemical evidence when it cannot be otherwise assailed. In Reg. v. Tawell (Bucks Lent Assizes, 1845), the prisoner was indicted for the murder of a woman named Sarah Hart. The deceased was found insensible and dying, and no accurate account of the symptoms could be obtained, as no one but the criminal was present. The body was examined eighteen hours after death, but no odor was perceived about the mouth, or in some blood which had been drawn from the body. The lungs were slightly congested, and there were some old pleuritic adhesions, but there was no disease of any organ to account for death. The stomach and bowels presented no morbid change. The contents of the former amounted to twelve ounces of liquid, having no odor of prussic acid, but merely a strongly acid smell of beer. (Lancet, April 5, 1845, p. 379; Northern Journal of Medicine, May, 1845.) They consisted of partially digested food, intermixed with the pulp of apple without the pips. Prussic acid was obtained from the contents of the stomach by distillation: it was identified by the application of the usual tests, and after separation as cyanide of silver, by its odor. The quantity thus obtained amounted to one grain of anhydrous acid, equal to *fifty minims* of pharmacopœial acid. The administration of the poison to the deceased was clearly brought home to the prisoner, partly by a series of moral circumstances of a most convincing kind, and partly by his own admissions. He attributed death to suicide; but this was entirely out of the question.

The case is chiefly remarkable from the medical assumptions made by the learned counsel who conducted the defence. They were,—1. That there must be direct evidence of death from poison. 2. That the evidence of medical witnesses who had never before met with a case of poisoning by prussic acid should not be received. 3. That the *quantity* of poison found in a stomach should be sufficient to cause death! "If there was not enough prussic acid in the stomach to account for death, there was an end of the case." (See on this fallacy, p. 184.) 4. That although a *grain* of prussic acid (anhydrous) was there found, yet the witnesses had not proved *upon their own knowledge and experience*, that this was sufficient to cause death! 5. That because persons had recovered from a larger quantity than was found in deceased's stomach, it was not to be inferred that she had died from the effects of a grain. 6. That because the odor of prussic acid was not perceived in the stomach before distillation, there could have been none present at the time of inspection; and that prussic acid could not destroy life in any case without leaving the odor behind it. 7. That a grain of anhydrous prussic acid might have been *spontaneously produced* in the stomach of the deceased after death, partly from the *pips of apples*, a portion of cake, the saliva swallowed during mastication, and partly from the decomposition of animal matter! It need hardly be observed that this theory of the spontaneous production of prussic acid in the stomach was invented by the chemists retained for the defence, including among others Mr. Herapath and Dr. Letheby. Every scientific point involved in this defence was based either upon a fallacy or a misstatement. The learned judge (Lord Wensleydale) who tried the case, showed that he was fully competent to unravel and expose the errors, legal and medical, involved in such subtle and dangerous propositions. The statement of the prisoner's counsel, that it was a rule [illegible] aw that there should be direct proof of death having been caused by poison, and of the presence in the stomach of a sufficient quantity of poison to produce death, was not true, neither was it necessary to prove what quantity of prussic acid would destroy life by the testimony of a person who had actually seen a human life destroyed by it. With regard to the smell, the only conclusion from the evidence was, that smell was a proof of the presence of the poison, but that the absence of smell was no proof of its absence. According to the witnesses, a grain, or even less than a grain, of prussic acid taken into the stomach, was sufficient to cause death. With respect to the allegation that prussic acid might be obtained from apple-pips, Mr. Cooper, the chemical witness, found apple but no pips in the stomach, and it was only by the distillation of the (bruised) pips that the acid was formed. After a short deliberation the jury returned a verdict of guilty, and the prisoner, before execution, confessed that he had perpetrated the murder.

An opinion has gone abroad, that the poison, in this case, was not administered by the mouth, but in a more secret manner. This opinion was based on the absence of odor in the stomach, and on the reported confession of the criminal. It was, however, founded on a mistaken view of the medical facts. One grain of anhydrous prussic acid was certainly found in the stomach, and this dose was of itself sufficient to cause insensibility and rapid death.

It was only by presuming on a general want of information regarding poisons among barristers, that such an unsound defence as this could have been suggested. In an experiment I found that the seeds of ten common apples distilled with water (the husks of the seeds being unbroken) did not yield in the distillate the slightest trace of prussic acid. When they were bruised and redistilled in a raw state (unboiled), there was a mere trace of prussic acid in the distilled liquid, and a quantity of Prussian blue, equal to the 1-150th grain, was obtained. (See Med. Gaz. vol. xxxvi, p. 328.) (See p. 627.) Such a pseudo-scientific theory as this to account for the presence of a fatal dose of prussic acid in a dead stomach will probably never again be brought forward. The odium of it has, most undeservedly, fallen on the lawyer who made use of it, in place of the chemists who suggested it, and who must have known that it was wholly inconsistent with the medical facts of the case.

In the case of Montgomery (Glasgow, Dec. 1857), the views of Orfila and other chemists, regarding the alleged production of prussic acid in a dead body were strongly urged in the defence. Only traces of prussic acid were found in the stomach after the long period of seventeen days, and in the preserved spleen nearly two months after death. The late Lord Justice Clerk, in addressing the jury, dismissed this theory as wholly improbable and unsustained by any facts. "If it were true, this acid," he said, "would be found in the body not only in cases of poisoning, but in many other cases. He trusted that it might never be again brought forward in the hope to perplex and mislead a jury, and to try to take off the effect of the clear and decided proof of the existence of poison in the body, and of the possession and use of poison by the accused." (Cowan's Report, p. 100.)

The poison not detected.—A person may die from prussic acid and none be found in the body. Assuming that a small but fatal dose has been administered, and that the dead body has been exposed or buried for a few weeks, it is not probable that any of the poison would be found by chemical analysis. The odor may entirely disappear in a week, and the longest period at which the poison itself has been found in the body is seventeen days. In this case the quantity was so small that no attempt was made to estimate it (case of Montgomery, supra), while the dose which was actually taken was four times as large as that which has sufficed to destroy the life of an adult. If one-fourth of the quantity had been taken, it is probable that not a trace would have been found. Prussic acid is rapidly lost by its great volatility. (See p. 182.) Unless, even in a recent inspection of the body, the viscera are placed in stoppered bottles, the whole may disappear within a few hours or days. These remarks apply to the blood and tissues with even greater force than to the stomach; for the quantity retained as a result of the absorption is generally small, and is constantly diminishing, unless the viscera are closely sealed up.

Supposing that neither by distillation nor by evaporation of the contents of a stomach, any prussic acid or any product of its decomposition (sulphocyanate) is obtained, then it is clear that there is none in the body; for it is not probable that any would be found in the tissues or blood, if none were contained in the stomach. Still, for reasons elsewhere stated, it must not be assumed that death has not been caused by prussic acid. The symptoms, the mode of death, and the moral circumstances of the case, may supply the evidence necessary for conviction. (See case of Walter Palmer, p. 597.)

On the other hand, prussic acid may not be obtained from the putrescent contents of the stomach, and yet they may contain an alkaline sulphocyanate, or a substance possessing its chemical properties. In the former edition of this work, I had suggested that this conversion to sulphocyanate in the presence of hydrosulphate of ammonia, might account for the partial loss of prussic acid in the dead body. The following experiment was performed with a view to try this question. On the 30th of June, 1858, one drachm of prussic acid (of 8½ per cent.) was added to the viscera of a person who had died suddenly, but not from poison. They consisted of part of the stomach, liver, and intestines, contained in a jar, loosely covered by an earthenware cover. The acid was after mixture detected immediately by the vapor tests. On the following day (July 1), it required a period of six minutes for the detection of the vapor. On the 3d of July, the silver test failed to detect the vapor, and the sulphur test produced only a faint indication. As the acid could no longer be detected by the vapor, the contents of the jar were left to pass through all the stages of putrefaction at a temperature reaching sometimes as high as 80° or 90°, and they were not again examined until the 16th of October. The viscera were then highly offensive. The silver test indicated only sulphuretted hydrogen, and the sulphur test gave no trace of prussic acid in the vapor. The viscera were digested in a mixture of one part of alcohol to four of water, at a moderate heat, and after twenty-four hours the liquid was poured off and filtered. It gave a full and decided reaction of sulphocyanic acid, with a persalt of iron, and this acid was found to be in combination with ammonia. This result rendered it highly probable that some portion of the prussic acid had become converted in the putrescent viscera to sulphocyanic acid, and that traces of the poison were thus found after *three months and a half*. Considering, however, that an alkaline sulphocyanide is a constituent of saliva, and that sulphocyanogen may be a product of decomposition, it would not be proper, from a solitary result, to infer in another case that prussic acid must have been in the stomach at the time of death. In Mr. Herapath's case (page 618), he failed to find the sulphocyanic acid in the *stomach*, but he states, that he found it in a small quantity of *blood*. Considering that the quantity of this poison which enters the blood is at all times exceedingly small, and that it is rapidly evolved from it, it is not at all probable that the acid would remain in the blood to be fixed by putrefaction for a longer time than it would remain in the stomach. On the contrary, as death is rapid, it is in the contents of the stomach, if anywhere, that we must look for this conversion. The non-discovery of sulphocyanic acid in the contents of the stomach, and the non-detection of the essential oil of almonds (the poison alleged to have been taken) in any part of the body, render Mr. Herapath's case one of mere speculation. At present we must look for conclusive and satisfactory evidence only in the detection and separation of prussic acid itself.

Quantitative analysis.—It is often a matter of importance in reference to fatal dose, the identity of a particular acid, &c., to ascertain the strength of the prussic acid taken. It is much more satisfactory to determine this point by chemical processes than by giving the poison to dogs or rabbits, and noting how long a time it requires for a certain dose to destroy life, or by assuming its strength from its designation. A measured and weighed quantity of the acid may be precipitated entirely by a solution of nitrate of silver. The precipitate should be washed and dried in a water-bath until it no longer loses weight. One hundred grains of dry cyanide of silver are equivalent to 20·14 grains of anhydrous prussic acid; this is in the proportion of about one-fifth, so that the weight of the dried cyanide divided by five, gives, with sufficient accuracy for common purposes, the quantity of anhydrous prussic acid present. One hundred grains of the *London pharmacopœial acid* should therefore yield ten grains of cyanide of silver: and the same quantity of *Scheele's acid*,—from twenty to twenty-five grains. The presence of hydrochloric acid, either as the result of distillation or of direct addition, may give rise to a fallacy in the estimation of the quantity of prussic acid. In order to guard against this, the acid or the distillate may be redistilled with finely powdered borax or chalk, before the precipitation by nitrate of silver. The foreign acid will be thus separated from it.

CHAPTER XXXVIII.

SUBSTANCES PRODUCING PRUSSIC ACID—BITTER ALMONDS—ESSENTIAL OIL—SYMPTOMS—APPEARANCES—FATAL DOSE—PERIOD OF DEATH—TREATMENT—ANALYSIS.

BITTER ALMONDS—THE ESSENTIAL OIL.

THE bitter almond owes its poisonous properties to prussic acid, which is easily obtained from it in a state of admixture with an essential oil, by distillation with water. It is, however, a remarkable fact, that none of the acid exists ready formed in it, and the poison is not produced except by the agency of water on the almond pulp. Heat is not required for this reaction; the mere trituration of the pulp with cold water is sufficient to produce the acid. Several cases are reported by Wibmer, in which serious symptoms occurred in children who had eaten immoderately of bitter almonds. (Arzneimittel. Amygdalus.) A girl, æt. 5, was nearly killed by eating a portion of bitter almond cake. M. Bonjean relates that a cow was poisoned by drinking water into which a small portion of the residue left after the expression of the fixed oil had been put. (Faits Chimiques, &c. p. 56.) There are two instances recorded in which bitter almonds are reported to have caused death in the human subject, but the facts are by no means clearly detailed. Judging from reported cases, a large quantity may be taken, even by children, without necessarily destroying life. Dr. Schlesier met with an instance in which a boy between two and three years of age, ate an ounce of bitter almonds (about 54). A quarter of an hour afterwards there was a general relaxation of the limbs; the countenance was pale, depressed, and drooping; the pupils dilated; respiration sighing; there was also a tendency to sleep, followed by vomiting of the coarsely digested pulp of the almond, which had a very strong smell of prussic acid. Emetics with ammonia, and exposure to a free current of air, soon restored him. (Canstatt's Jahresbericht, 1844, B. v, s. 289. See also Ed. Monthly Journal, Oct. 1850, p. 379.)

ESSENTIAL OIL. PEACH-NUT OIL.—The essential oil, which is produced by the distillation of the pulp of the bitter almond with water, is a powerful poison, and has caused numerous deaths. In 1837–8, there were only four deaths recorded from poisoning by this oil. It is now, however, a frequent cause of death as a result of accident or suicide.

Its taste and odor render it difficult of administration for the purpose of murder; nevertheless, the case of Reg. v. Fisher (York Lent Ass. 1855), shows that it may be thus used. In this case there was a suspicion that it might have been poured down the deceased's throat while he was asleep. Its poisonous properties are due to the presence of prussic acid, which is intimately combined with it. Five pounds of the almonds are calculated to yield about half an ounce of the oil, and the quantity of anhydrous hydrocyanic acid contained in it, varies from eight to fourteen per cent.—on an average ten per cent. I find, by another calculation, that 2500 parts of bitter almonds yield 100 parts of amygdaline, and these by a reaction with the elements of water, produce 41 parts of essential oil and 6 parts of anhydrous prussic acid: hence 100 grains of bitter almonds (equal to ten in number) would be equivalent to 1·88 grains of essential oil, and 0·24 grains of anhydrous prussic acid. One hundred parts of the essential oil w[illegible]tain 12·76 parts of anhydrous prussic acid, and it would require [illegible]ns of bitter almonds to represent 100 grains of the prussic acid of the London Pharmacopœia. This oil must, therefore, be regarded as a [illegible] active poison, being at least four times as strong as the pharmac[illegible]l acid, but it becomes weaker by keeping. Its uncertain strength renders it unfit for internal use: but in France it is given in doses of from one-quarter of a drop to a drop. The oil is sold to the public in quantities of not less than a quarter of an ounce, at the rate of from three to five shillings per ounce. The liquid called ALMOND FLAVOR, spirit of almonds, or essence of peach-kernels, contains one drachm of the essential oil to seven or eight drachms of spirit. It is sold to the public in quantities of not less than a quarter of an ounce, at the rate of one shilling per ounce, for the purpose of giving a pleasant flavor to pastry! It may be as well to state that one ounce of this almond flavor is at the lowest computation equivalent in strength to 250 grains of the pharmacopœial prussic acid. In some cases it may happen to be nearly equal in strength to this poison, and yet it is sold without restriction, and it is intrusted in private families in the hands of ignorant cooks to apportion the dose which may give the requisite flavor to food!

It is estimated that about 8000 pounds of the ordinary essential oil of almonds are annually manufactured for the retail British trade. Since the introduction of the cheaper nitro-benzole, which has a similar odor and flavor, into the manufacture of perfumery, the whole of the above large quantity is chiefly disposed of for the purpose of eating and drinking! (Lancet, Jan. 10, 1857, p. 46.)

Effects of the vapor.—The odor of the oil is not derived from prussic acid, as it is equally powerful and possesses the same character whether prussic acid is present or not. The vapor of the acid is not so freely evolved from the oil as from the watery solution. A question respecting the poisonous properties of the vapor arose in the subjoined case, which occurred in London, in 1838. The deceased, the wife of a publican, had been clearing out a closet, which contained, among other liquors, a bottle of the essential oil of bitter almonds. She was suddenly heard to call out. A servant found her pale and faint, and she complained of sickness. There was a strong odor in the room, and deceased said that the corks of some of the bottles had come out, and the smell had made her feel sick. She was removed to bed, but died before any medical assistance could be obtained. There was no motive for the deceased committing suicide, and it was a subject of inquiry, whether the *vapor* alone might not have caused her death. This question was set at rest by an inspection of the body. Some of the poison was found in the stomach, and there was a very strong odor of bitter almonds in the contents. It was, therefore, clear that the deceased must have swallowed a portion of the poison; whether from motives of curiosity or not, it is impossible to say. The medical witness, in answer to a question, properly stated, that less than a teaspoonful might cause the death of an adult. The vapor may produce giddiness and stupor; but unless long respired, it would not be likely to cause fatal effects.

Symptoms.—The following case by Mertzdorff will illustrate the effects produced by this poison. A hypochondriac, æt. 48, swallowed two drachms of the ethereal oil of bitter almonds, and immediately threw himself on his bed. In a few minutes afterwards, having spoken to an attendant in the room and asked for water, his features became distorted, his eyes were turned upwards, fixed, and projecting from their sockets. His chest heaved violently and rapidly. A medical man arrived in about twenty minutes after the poison had been swallowed, and found the patient quite unconscious, his eyes open and staring, the pupils immovable, the respiration slow, prolonged, and accompanied with a rattling noise in the throat. The pulsation of the radial and carotid arteries, as well as of the heart itself, could scarcely be perceived, and followed each other every two seconds (30 per minute). Swallowing was no longer possible: a strong odor of bitter almonds issued from the mouth. In ten minutes more the patient was dead, making the duration of the case half an hour.

In a case, the particulars of which were communicated to me by Dr. Bull, of Hereford, a woman swallowed about seventeen drops of the essential oil, and she died in half an hour. She was seen by Dr. Bull in about fifteen minutes: her face was livid; the lips were separated; the teeth clenched; there was froth about the mouth; the eyes were half shut and glassy; the pupils dilated and fixed; there were heavings of the chest at intervals; there was no pulse, and the action of the heart was scarcely perceptible. No odor was perceived about the body until after the stomach-pump had been used. The first symptoms observed in this case were strong convulsions,—the deceased throwing her arms about her as if in pain. A boy, æt. 13, swallowed a quantity of the oil; he was found lying on the floor motionless and insensible; his face pale; his eyes open and fixed,—the pupils dilated, and he was rolling and panting for breath; the pulse at the wrist was imperceptible; he died in a quarter of an hour without any convulsions appearing. A man, æt. 20, swallowed about two ounces of the oil. A person present saw him fall suddenly while in the act of swallowing,—he made a loud cry, gave one deep expiration, and died.

In another case, a woman, æt. 46, who had been in the habit of using the almond essence for flavoring confectionery, swallowed about half an ounce (thirty drops of the oil). She died in less than half an hour.

When seen by a medical man ten minutes after she had taken the poison, she was perfectly insensible. The face was pale but swollen, and covered with perspiration; the eyes stared fixedly as if in terror; the pupils were dilated. The lips were partly closed and livid, and a frothy mucus issued from the mouth. The lower jaw was firmly contracted, while the muscles of the neck and of the limbs, except those of the fingers, were flaccid. She breathed slowly and heavily, making about ten respirations in a minute; the pulse was from 30 to 40, and feeble. There was an odor of bitter almonds in the breath. Some blood which was drawn from the arm was thick and dark, resembling choleraic blood. In spite of the use of the stomach-pump and cold affusion, the patient did not show any signs of recovery, but gradually sank. (Assoc. Med. Jour. Dec. 13, 1856, p. 1055.)

The following may be taken as a summary of the symptoms: lividity of the face; eyes glassy, prominent, fixed and staring; pupils dilated and insensible to light; jaws spasmodically closed; frothy mucus about the mouth; in some cases, vomiting of food; coldness of the skin; heaving and intermittent respiration, sometimes stertorous; absence of the pulse; head spasmodically drawn backwards, and sometimes the trunk; general relaxation of the limbs; an odor of bitter almonds about the mouth.

The symptoms have been known to commence in the act of swallowing the poison, or, as in Mr. Chavasse's case (p. 613), in half a minute; but, as in poisoning by prussic acid, a person may retain for a certain period consciousness and a power of performing certain acts. The following case, which occurred to Mr. Wakefield, is in this respect instructive. A boy, æt. 19, was sentenced by a police-magistrate to imprisonment. At twenty minutes before six P.M. he was put into the prison-van in perfect health; he reached the prison at a quarter after seven, and here, on leaving the van, he attracted the notice of the gate-keeper, who called him by his name: he answered feebly: "That's me, that's me." With slight assistance he walked to the room where the prisoners are searched, when a half-ounce bottle was found upon him, labelled "Essential Oil of Almonds." It was then suspected that he had taken poison, and Mr. Wakefield was sent for. The boy stood in the room erect for five minutes. There was nothing striking or peculiar in his appearance, no smell of poison at the mouth; the pupils were dilated; the pulse was rapid and feeble; the skin pale. The stomach-pump was used, and the smell of the oil was perceptible in the liquid extracted. Ammonia was administered, and warmth applied to the limbs, but he expired just three hours after he had entered the van in perfect health. It was ascertained that the bottle had contained two drachms of the oil. (Lancet, Dec. 13, 1845, p. 656.) This case is remarkable in two points of view, considering the large dose swallowed: 1. The length of time which elapsed before well-marked symptoms of poisoning appeared; and, 2. The length of time which the deceased survived after their appearance. In Dr. Bull's case (supra) there was a short interval between the taking of the poison and the production of insensibility. The deceased called out, and she had time to cork a small bottle which had contained the poison, to put it into a bag, draw the strings of the bag, and hang it over a chair by the side of the bed. A man, æt. 50, swallowed six drachms of the oil of bitter almonds. He walked down stairs after taking the poison, spoke to his son, and lived about ten minutes. In this case, the lungs were remarkably emphysematous, the air-cells being distended into bladders. The heart was full of blood, and the foramen ovale open. (Harveian Society, Lancet, Jan. 30, 1858, p. 128.)

In October, 1845, a case was referred to me for examination by Mr. Savage, in which there was also clear evidence of the power of locomotion after probably a large dose of this poison had been taken. The deceased mixed the poison with some ale in a cup, stirred it up with a pipe, and drank off the greater part. Five minutes had elapsed when he was seen deliberately walking towards a staircase apparently conscious and self-possessed, for he replied rationally to a question put to him. The symptoms then came on very suddenly, and commenced with vomiting, during which, probably, part of the oil which he had swallowed, was ejected. He became insensible; the breathing was convulsive and took place at intervals; but, excepting slight opisthotonos, there were no convulsions. From the facts observed by Mr. Savage, it appears probable that the whole duration of this case did not exceed seven minutes; and the fatal symptoms were not manifested until within the last two minutes.

In March, 1853, Mr. Phillips of Coventry communicated to me the following case: A woman, æt. 39, swallowed half an ounce of almond flavor, containing half a drachm of the essential oil. After swallowing the poison, she seized a water-jug, walked to a tap in the yard, and drew and drank off a large quantity of water. She then went up two flights of stairs, calling for her child, descended one flight to a bed-room, and was heard to fall heavily on the bed. These acts occupied about *five minutes.* In another five minutes she was seen by Mr. Phillips, who found her perfectly insensible and motionless; the pupils were moderately dilated and insensible to light; the mouth was partly open, the lips were pale, there was no distortion or spasmodic movement of the features; the pulse was slightly tremulous, entirely ceasing in a few minutes; the breathing slightly stertorous, and taking place at long intervals. She continued in this state for twenty minutes without the least convulsive movement of the body, when she died, *i. e.* half an hour after she had taken the poison. There was a strong odor of bitter almonds in the room.

Appearances after death.—In Mertzdorff's case (p. 607), which proved fatal in half an hour, the body was examined twenty-nine hours after death. There was discoloration of the surface, and decomposition had advanced considerably. There flowed from the nostrils and mouth, each time the body was moved, a bloody-looking fluid, which, as well as the whole body, but especially the internal parts, emitted a powerful odor of bitter almonds: the odor was so strong as to conceal even that resulting from putrefaction. It was perceived most intensely on opening the cavity of the abdomen. In the stomach were found about six ounces of a brownish fluid which possessed the odor of bitter almonds in a marked degree. The internal surface of this organ, as well as of the small intestines, was considerably reddened. The smell of bitter almonds in the intestines was less obvious the further they were examined from the stomach. There was congestion of the vessels of the brain. The blood generally was fluid, and, as well as the bile and muscles, of a dark purple or violet color. No appearance worthy of notice was observed in any other part of the body. (Horn's Archiv. für Mediz. Erfahr, 1823, B. ii, s. 60. An abstract of this case will be found in the Ed. Med. and Surg. Jour. xxii, 332.) In the case which occurred to Dr. Bull (p. 607), fatal in half an hour, on inspection nine hours after death no odor of almonds was perceptible in the cavities of the chest, head, or heart, nor in the venous blood with which the system was gorged. The lungs and heart were healthy. The vessels of the brain were congested, and there was a general effusion of serum on the hemispheres. The lining membrane of the stomach was much congested. On opening it the bitter-almond odor was quite perceptible. (See Prov. Med. Jour. Sept. 11, 1844, p. 364.) In the case of the boy (p. 607), which proved fatal in a quarter of an hour, on inspection there was pallor of the face, with lividity of the depending parts; the lungs were congested; the odor of the poison was perceptible only in the abdomen, and very distinctly in the contents of the stomach. The mucous coat of this organ was generally pale, but there were some patches of ecchymosis scattered over it. The essential oil and prussic acid were detected in it. (Lancet, July 12, 1845, 40.) In the body of the man (p. 607) who fell suddenly while in the act of swallowing a large dose of the poison, and died after a deep expiration, the following appearances were met with. A large quantity of the poison was found in the stomach, and the smell of bitter almonds was perceptible in the brain. The venous system was filled with dark liquid blood. The lungs were healthy. (Canstatt's Jahresbericht, 1844, v, 290.)

After death, even from a large dose, the odor is not, however, always perceptible about the body. A case of poisoning by the oil of bitter almonds occurred at Hornsey, in February, 1843, which shows that an inspection is absolutely necessary in order to determine the cause of death. A chemist was found one morning lying dead on the floor of his shop. A surgeon who was called a few hours afterwards, suspected that the deceased had taken poison, because he saw on the shelf near the body a bottle which had contained the essential oil of bitter almonds. There was, however, no odor about the mouth, and this led to the erroneous opinion that the deceased had died from disease of the heart. The body was subsequently inspected, and it was soon rendered evident, by the powerful odor which escaped from the cavities, that the deceased had died from the effects of the oil of bitter almonds. All the viscera were in a healthy state. (Med. Gaz. April 7, 1843.)

In Mr. Wakefield's case (p. 609), that proved fatal in three hours, the skin was partially livid, the blood fluid and the membranes of the brain as well as the lungs were gorged. The contents of the stomach had a strong smell of the oil, and the mucous coat towards the intestinal opening had a red appearance. The other organs were healthy. In Mr. Savage's case (p. 609), which proved fatal in seven minutes, the countenance was pale; cadaveric rigidity (eighteen hours after death) was strongly developed; and the hands and feet were unusually livid. The eyes were remarkably brilliant. The blood, with which the venous system was gorged, was liquid and of a dark color. The lungs and heart were healthy. There was an odor of the oil of bitter almonds on opening the chest, rendering it probable that either a portion of the essential oil, or its odoriferous principle, had been absorbed or diffused throughout the body. The brain, which was perfectly healthy and free from congestion, gave out a slight odor of bitter almonds. The mucous membrane of the stomach was reddened from inflammation about the cardiac extremity and œsophageal opening. When I saw it (ten days after death), although the stomach had probably undergone some change, the cardiac extremity of the organ appeared as if it had been acted upon by a powerful irritant. It would thus appear that the essential oil of bitter almonds, which has a hot and burning taste, like some other essential oils, may exert an irritant action when taken in a large dose. Prussic acid was detected in the contents of the stomach, and the odor of bitter almonds was very powerful in this organ so late as *ten days* after death.

In Mr. Phillips's case (p. 609), which proved fatal from a small dose in half an hour, on inspection twenty-four hours after death, there was a strong smell of bitter almonds at the mouth. The vessels of the brain were much congested with dark fluid blood, and the odor of the poison was perceptible in the brain on making a section through it. The lungs were gorged with black blood throughout. The left ventricle of the heart was firmly contracted,—the right cavities were moderately distended. The stomach contained four ounces of a fluid smelling strongly of bitter almonds; its mucous membrane presented a slight inflammatory redness towards the greater end. The intestines were healthy. There was no congestion of the liver, spleen, or kidneys. Mr. Phillips forwarded to me for examination the contents of the stomach, and about two drachms of blood taken from the right auricle of the heart. The contents of the stomach, amounting to about three ounces, were reddish colored, owing to the presence of a small quantity of blood: they were liquid, and slightly acid. When the examination was made, *i. e. five days* after death, they had a slightly aromatic smell; but an experienced person unacquainted with the facts could perceive in them no odor of prussic acid or of bitter almonds. Prussic acid was, however, detected in the vapor issuing from the liquid by the silver and sulphur tests: and by distillation a quantity of colorless liquid was procured, from which cyanide of silver and Prussian blue were readily obtained. The blood was of a dark red color. It had separated perfectly into serum and coagulum, like healthy blood. It had no particular odor; and neither before nor after distillation did it yield any indication of the presence of the poison. When examined under a microscope, with a power of 300 diameters, the corpuscles were seen of their usual size and shape: they had undergone no physical change. (This is adverse to the retention of prussic acid, as suggested by Mr. Herapath, see p. 604.)

In May, 1855, a woman, æt. 24, who had swallowed a drachm of the essential oil, was brought to Guy's Hospital, and died shortly afterwards. About twenty minutes previous to her admission, she had been seen walking about, staring with her eyes, and in a very excited state. Soon afterwards she was found lying in a passage in a state of insensibility.

When admitted, her face was livid, and her eyes were injected and starting from her head. A few inspirations were made, but with the greatest difficulty, and she then expired. Cold affusion and other treatment merely produced convulsive motions of the chest, with an escape of frothy mucus from the mouth and nostrils. The body was examined four and a half hours after death. The muscles of the head and arms were flaccid, while those of the legs were rigid. The face was bloated, and of a dark purple color, presenting the appearance seen in some cases of asphyxia. The eyes were partially open, full, and glassy, and the conjunctivæ between the eyelids were congested. The pupils were slightly dilated. At this very early period for an examination there was no odor about the body! The brain was remarkably congested, the spinal cord was healthy. The lungs presented bright round ecchymosed patches; the mucous membrane of the air-passages was reddened. The right cavities and large venous trunks of the heart were much distended with dark fluid blood. The left auricle contained a small quantity, the left ventricle was empty and contracted. The tissue of the heart was flaccid, and the organ generally flabby. The blood had a doubtful odor of bitter almonds. The stomach contained a pint of brown curdy fluid, having a strong odor of bitter almonds. The mucous membrane was covered with a thick layer of mucus; it was acutely inflamed, highly injected with blood, and of a bright pink color. This was especially the case at the cardiac (gullet) end, and the last two inches of the gullet were of a bright pink hue. The membrane of the duodenum and the upper eight feet of the small intestines had also a pink hue: the serum presented this pink color, but in a smaller degree. Dr. Odling detected prussic acid in the distilled contents of the stomach, and on the fourth day, I detected traces in a portion of the undistilled contents by the vapor tests. The blood which was not putrefied had no odor of prussic acid or of the essential oil. Dr. Odling did not detect any trace of the poison in it, and my examination of another portion on the fourth day led to only a doubtful result. (Guy's Hosp. Reports, 1854, p. 367.) This is an additional fact adverse to the hypothesis of the retention of prussic acid by blood (see p. 604).

In the case of Mr. Sadleir (Feb. 1856), whose death was caused by a very large dose, there was a strong odor of the oil at the mouth, but no froth; the eyes were lifelike and glistening, the pupils dilated. The body was examined forty-eight hours after death. There was congestion of the lungs and bronchial tubes. The right auricle of the heart was distended with blood; the other cavities were empty. The odor of the oil was perceptible throughout the body. The stomach contained ten ounces of undigested food, mixed with from two to three ounces of the oil. There was slight congestion of the intestines. The kidneys, as well as the brain and its membranes, were congested. There was an effusion of bloody serum at the base of the brain. In the case of a girl, æt. 18, who died in a few minutes from a dose of almond flavor, equivalent to about thirty drops of the oil (communicated to me by Mr. Hunt, of Bath, June, 1856), the appearances were much the same; there was engorgement of the lungs, with distension of the cavities of both sides of the heart. The blood was fluid. From this it will be perceived that the appearances in the body are by no means uniform. There is commonly a general congestion of the organs with dark liquid blood and an odor of the poison throughout the cavities. In some cases the congestion is most marked in the brain, in others, in the lungs and heart, and in others, again, in the viscera of the abdomen.

Fatal dose.—The essential oil of bitter almonds varies much in strength. The prussic acid contained in it may amount on an average to ten per cent.; but it may be procured free from prussic acid. Sir B. Brodie, with the design of tasting it, applied *one drop* to his tongue. He immediately felt a remarkable and unpleasant sensation in the pit of the stomach, with such weakness in the limbs and loss of power in the muscles that he thought he should have fallen. (Paris, Med. Jur. ii, 404.) This proves that it is dangerous in the smallest doses, and quite unfit for medicinal use. The smallest quantity of the oil which has yet been known to destroy life was in the case which occurred to Dr. Bull of Hereford (p. 607). A woman, æt. 49, was in this instance killed in half an hour by a dose of less than twenty drops. Probably, not more than *seventeen drops* were taken. A dose of half a drachm, or thirty minims, has destroyed the life of an adult. In a case which occurred to Mr. Chavasse, a man recovered from a similar dose, but with some difficulty. A druggist swallowed half an ounce of almond flavor (equivalent to thirty drops of oil). He was seen ten minutes afterwards. In less than half a minute after he had swallowed the poison, he fell down in a state of syncope, his face was deadly pale, and his pulse (according to a bystander) quite imperceptible. After a few minutes he came to himself, and he was then put to bed. The moment he recovered from the fainting, he vomited some undigested food and bile, strongly impregnated with the odor of prussic acid (oil of almonds ?). He then became delirious, muttering to himself, and speaking almost incoherently. In a short time the delirium ceased; the whole frame was slightly convulsed; but the convulsions in a minute or two ceased, excepting in the eyelids. For a few minutes he was sensible, and spoke on the nature of his attack, but again gradually relapsed into a delirious state; his face had an expression of excessive joy; his eyes shone brilliantly; indeed, he exhibited the appearance of one who had been inhaling the laughing gas. These symptoms continued for a few minutes; he again became sensible, and expressed himself as being much better. The pulse, which was before quick and intermittent, now became more slow and regular; the expression of the face assumed a more natural aspect, with the exception of the eyes, which throughout the attack continued extremely brilliant. While the attack lasted, the respiration was very short, and when the patient was conscious he felt as if he should be suffocated. The surface of the body during the whole time was cold. He gradually recovered from the effects of the poison. (Lancet, Sept. 1839, p. 930.) This case is especially remarkable in the fact that, so far as I can ascertain, it is the only instance of a temporary *remission* of symptoms in this form of poisoning.

Considering the fatal effects produced by a dose of less than twenty drops, and the severity of the symptoms, from which a person only recovered under active and immediate treatment, after taking thirty drops, we may infer that a dose of from *twenty* to *forty drops* of the oil containing prussic acid may prove fatal to adults under common circumstances. Children would die from a still smaller quantity; nevertheless, there is a case on record in which a girl, æt. 9, recovered from a dose equivalent to seven drops. A girl swallowed about a teaspoonful of a mixture sold by druggists as "ratafia," composed of one part of the essential oil of bitter almonds to seven parts of spirit. The quantity swallowed by the patient was equivalent to about *seven drops* of the essential oil. When seen immediately after the accident, there was complete insensibility; the eyelids were closed, but the eyes were brilliant and glassy, without any mental expression; the pupils dilated; there was no pulse at the wrist; the carotids beat fully and quickly; there was relaxation of the muscles of the limbs, but the lower jaw was clenched in rigid spasm. Cold affusion with stimulants, stimulating frictions and emetics, were employed. Vomiting was induced, and the evacuations had a strong smell of oil of almonds. In about twenty minutes the pulse returned,—the child opened her eyes, and was able to answer questions. This case shows that a small dose of the oil may give rise to alarming symptoms; and it is probable, that but for the active and prompt treatment adopted, the child would have died. (Lancet, June, 1844.)

A case occurred at Guy's Hospital in May, 1843, in which a boy, æt. 12, recovered under early treatment. The boy was accosted in the street by another boy, who had a medicine basket on his arm, and from this he took a bottle and offered to the patient some liquid which he called almond oil. He thought that he swallowed about a tablespoonful; he experienced shortly afterwards a burning sensation in his throat, and in about ten minutes he sat down on a door-step, and became insensible. In about eight minutes afterwards he was brought to the hospital. His breath smelt strongly of bitter almonds; there were violent tetanic convulsions, with complete opisthotonos; the head and neck being drawn backwards, the elbows were drawn behind his back, and firmly fixed in this position. The jaws were quite fixed: there was complete insensibility, and the pulse was scarcely perceptible. The treatment consisted in cold affusion to the spine and the use of the stomach-pump. The stomach was well washed out with a large quantity of water: the washings smelt strongly of bitter almonds. During the treatment, the patient suffered from strong convulsive twitchings of the muscles. In about an hour he recovered, and in the course of a few hours left the hospital. It is very probable that in this case the boy swallowed a portion of what is called almond flavor, a diluted solution of the essential oil. The contents of the stomach were submitted to two distillations, and about four ounces of a clear liquid, smelling strongly of bitter almonds, were procured. This liquid was scarcely rendered cloudy by nitrate of silver, and the iron test gave no trace of Prussian blue. The only proof, therefore, of the nature of the poison, was, the odor of the essential oil, which was very powerful, notwithstanding the want of action in the tests.

The largest dose from which there has been recovery was in the following case. A boy, æt. 4, swallowed from a bottle about four or five drachms of the concentrated oil. He replaced the bottle on a table and ran out of the room. He then staggered and fell in a state of insensibility. In five minutes he was seen by a medical man, and he was then laboring under the following symptoms: Countenance flushed—eyeballs prominent and protruding from their sockets with a rolling motion,—pupils widely dilated and insensible to light,—pulse full and strong, but slow,—breathing stertorous,—complete opisthotonos and frequent convulsive action of the muscles of the face and neck. Cold affusion and the stomach-pump were immediately employed, and the child recovered in about two hours. (Lancet, Jan. 13, 1855, p. 84.)

In the following case a woman recovered, but the quantity of almond flavor taken by her is not stated: it was, however, sufficient to produce serious symptoms. When seen ten minutes after she had taken the poison, the patient was quite insensible: her countenance was ghastly—the eyes were fixed, staring, and prominent,—the pupils dilated,—the jaws firmly closed,—a frothy mucus exuding from the mouth: the breathing was heavy and stertorous: the pulse at the wrist was scarcely perceptible, but the carotid arteries pulsated strongly,—the limbs were relaxed. There was an odor of bitter almonds. Cold affusion was resorted to: ammonia was applied to the nostrils, camphor liniment to the chest, and mustard poultices to the legs. This treatment was persisted in for two hours, and there were then convulsive twitchings, with rolling of the eyes. In another hour the patient was delirious; she then fell into a sleep and ultimately recovered. (Brit. Med. Journal, May 8, 1858, p. 370.)

The largest dose of this poison recorded to have been taken was in the case of Mr. John Sadleir (Feb. 1856). He procured half a pint of the oil, and it is supposed that he made the attempt to swallow the greater part of this. Two ounces of the oil were found in his stomach after death.

Period of death.—This poison may destroy life with the rapidity of a strong dose of prussic acid. In one case, a man fell while in the act of swallowing the oil, and died instantly. In Mr. Savage's case, death took place in seven minutes. In the greater number of fatal cases, death has occurred within half an hour. Mr. Wakefield's case was unusually protracted, as the patient survived *three hours.* Like prussic acid, it either destroys life rapidly, or the person recovers.

Local action.—The essential oil of bitter almonds may, by the prussic acid contained in it, exert a local action. In small quantities it would act as a sedative; but, from its greater strength, its operation would be *cæteris paribus*, more violent than that of prussic acid. It is proper to mention, that this oil is employed in the preparation of numerous cosmetics which are applied to the skin. These cosmetics, which are extensively sold by perfumers, are to be regarded as highly poisonous compounds, the use of which for external application should be strictly prohibited. Local paralysis might easily arise from their employment. If applied to an abraded surface death might follow.

Treatment.—The treatment of a case of poisoning by the essential oil of almonds is the same as that directed for prussic acid (p. 587). If the case is seen early and the patient is not in a state of collapse, the stomach-pump may be used with benefit, the use of this instrument being continued until the liquid withdrawn has lost the odor of bitter almonds.

CHEMICAL ANALYSIS.

The *bitter almond* is readily known by its shortness, thickness, and ovoid shape, as well as by the taste and odor of its pulp. The pulp obtained from *one-half* of a bitter almond, bruised with cold water, faintly affected nitrate of silver by its volatility, but in a quarter of an hour gave clear evidence of prussic acid with the bihydrosulphate of ammonia, and other tests. The application of a gentle heat to the watch-glass containing the pulp of the almond, causes the vapor to be more rapidly evolved.

A question may arise respecting the quantity of prussic acid which may be produced in the human body from the bitter almond, as in the following case, which occurred in London, in November, 1844. A man was found dead, and there was a strong odor of bitter almonds in the room in which his body was discovered: there was also the appearance of froth about the mouth of the deceased. On inspection, some bitter almonds in an undigested state were found in the stomach. The quantity of prussic acid obtained by a distillation of the contents, was stated to be equal to one drachm of the common acid (P. L.). The medical practitioner is reported to have said, that this acid might not have been taken as such, but derived from the almonds. The anhydrous prussic acid obtained amounted to 1·2 grains, *i. e.*, quite enough to destroy life. The quantity of bitter almonds required to yield this, according to the calculation in the text (p. 606), would be about 500 grains (about 54 almonds). The weight of the almonds found in the stomach is not stated; but it is not likely that so many were eaten as to produce and leave in the stomach this large quantity of anhydrous prussic acid, nor would this account for the odor in the apartment. It is highly probable that the individual destroyed himself with prussic acid, having previously eaten some bitter almonds in order to conceal the odor of the acid. A case has been elsewhere related (ante, p. 605) in which a child recovered after having eaten an ounce.

The *essential oil*, which is often called peach-nut oil, is colorless when pure, but it commonly has a pale yellow color, and a strong odor of bitter almonds, by which it is at once identified. It has a hot, burning taste, and a feebly acid reaction. It gives a greasy stain when dropped on paper, which does not entirely disappear on the application of heat. It has a sp. gr. of 1·043: it sinks in water, which dissolves about one-thirtieth part. It is soluble in alcohol and ether in all proportions. When mixed with a few drops of strong sulphuric acid and heated, it forms a rich crimson-red liquid which, if exposed to air, acquires a yellow color. When poured into cold water, the crimson liquid is immediately destroyed, and a yellow coloring matter falls in globules. The smell and taste of this oil are sufficient for its identification: but nitrobenzole possesses the same odor, and may be mistaken for it.

The vapor of prussic acid does not so readily escape from this oil as from the watery solution: hence the vapor tests do not give the same characteristic results. *Tests.* 1. Add to one or two drops of the oil a like quantity of bihydrosulphate of ammonia. Mere mixture at a low temperature only produces sulphocyanate after standing ten minutes or longer: but if the liquid is warmed to 100°, the conversion is immediate (ante, p. 591), and the change is indicated by the blood-red color struck on adding to the liquid persulphate of iron. If any unchanged hydrosulphate should give a black color, this may be removed by the addition of one or two drops of hydrochloric acid. 2. Dissolve one or two drops of the oil in alcohol and add to the mixture a few drops of a solution of potash, followed by the solution of green sulphate of iron (ante, p. 591), and hydrochloric acid. Prussian blue is formed on agitating the mixture, but it does not appear until the precipitated oxide of iron is dissolved by the addition of diluted sulphuric or muriatic acid. The silver test is inapplicable to the oil in its ordinary state. The vapor of the oil produces no change in a drop of a solution of nitrate of silver, except after long exposure. If, however, the oil be heated, there is an immediate production of cyanide of silver. The two tests above mentioned are sufficient for all practical purposes.

Water will separate a small portion of prussic acid from the oil. Thus, by agitating in a tube about one drachm of the oil, with three or four drachms of distilled water, and after a few minutes, filtering through a *wet* filter, the oil is entirely separated. The liquid which passes through is scarcely acid; it is rendered cloudy by nitrate of silver, and gives a decided blue-colored precipitate with the sulphate of iron and caustic potash; it possesses all the properties of a weak solution of prussic acid. If the water be mixed with a little potash before agitating it with the oil, the acid is more effectually separated and may be detected at once by the iron test. The prussic acid may be separated more effectually from the oil by distilling it repeatedly over the finely powdered red oxide of mercury, or by agitating it with a mixture of milk of lime and protochloride of iron, and then submitting the mixture to distillation. The oil boils at 356° and is distilled at this temperature unaltered.

Organic liquids.—The odor of the oil would in general indicate its presence in any organic liquid. Owing to its great density the oil may be found at the bottom of the liquid while the prussic acid may be partly dissolved in the watery portion. The liquid may be distilled in the usual manner, and the oil and acid in the distillate examined by the tests above described. As ether readily dissolves the oil, this may be in some cases used as a medium for separating it. The oil has not been found in, or separated from the tissues, but it appears to undergo oxidation in the system. It is eliminated in the urine in the form of hippuric acid (ante, p. 87). In administering the oil deprived of prussic acid to rabbits, Dr. Maclagan found hippuric acid in the urine. (Pharm. Journal, Dec. 1853, p. 278.) In a case of alleged poisoning by this oil, Mr. Herapath states that he found no trace of prussic acid or of the oil in the body, but traces of sulphocyanic acid in the blood. (Chemist, 1854, p. 321.) I have elsewhere referred to this statement (ante, p. 604). So far as prussic acid is concerned, it is probable that in no case of poisoning does the blood receive by *absorption* at *one time* more than a minim to a pound: but if the proportion were a minim to an ounce, it is not probable (pp. 611, 612) that even this small quantity would remain in the blood to be converted during putrefaction to sulphocyanic acid, and detected as such two months after death! With a vagueness which deprives the result of all scientific value, the quantity of blood examined is not even mentioned: and the substance (hippuric acid) into which the essential oil is known to be converted was not even sought for.

The pure oil deprived of prussic acid.—This is known to chemists under the name of hydruret of benzyle. When deprived of prussic acid the energy of the oil as a poison ceases; but it still retains a noxious action on the animal system. It now requires to be given in larger doses, and its mode of operation is different. (Pharmaceutical Journal, July 1847, p. 11.) Dr. Maclagan found that a few drops of the oil deprived of prussic acid did not act as a poison on animals; but in larger doses, *i. e.* of a drachm and upwards, it was fatal to rabbits. Two drachms of the pure oil caused a rabbit to fall on its side in ten minutes, and it died in fifty minutes. (Pharm. Journal, Dec. 1853, p. 278.) Some experiments on the oil freed from prussic acid, by Mr. Langdale, are reported in the Lancet, Jan. 10, 1857, p. 45. One drachm given to a middle-sized dog, half a drachm to a cat, and four drops to a rat, did not destroy life, while four drops of the common oil destroyed a rat in two instances. In doses of from one to two drachms it has a tendency to cause inflammation of the stomach and bowels. Mr. Price Jones gave fifteen drops of the purified oil to a rabbit. The animal uttered a few cries, but recovered in about ten minutes. Thirty drops given to another rabbit produced violent convulsions with prostrations and oppressed breathing. The animal died in a minute and a half. Five drops of the ordinary essential oil killed a rabbit in about a minute. These facts viewed together show that the purified oil possesses noxious properties, although in a much smaller degree than the ordinary oil. Mitscherlich states that he found it still poisonous when quite free from prussic acid (Pharm. Jour. vol. x, p. 83); but it is a matter of great difficulty to deprive it entirely of the acid. For testing the purified oil we may agitate it with a weak solution of potash, filter through a wet filter, and then add sulphate of iron and hydrochloric acid, as in the use of the iron test for prussic acid. The production or non-production of Prussian blue will show the presence or absence of prussic acid. The sulphur test directly applied to the oil will equally reveal the presence of the poison.

Nitrobenzole or Nitrobenzine.—This compound of benzole and nitrous acid has the odor and flavor of bitter almonds in a most powerful degree. It is of a yellow color, sweet to the taste, boils at 434°, and has a sp. gr. of 1·209. It is insoluble in water, but soluble in alcohol and ether. It is used in perfumery and for cosmetics; it is said also to be used as a substitute for essential oil of almonds in flavoring confectionery. It is made from the rectified products of coal tar and nitric acid. One drachm of it killed a rabbit almost instantaneously. Half a drachm with two drachms of water rendered a cat insensible for several minutes, and a slimy mucus flowed from its mouth for several hours afterwards. It refused all food and died in twenty-four hours. (Lancet, Jan. 10, 1857, p. 46.)

In the use of poisonous compounds for flavoring food, it is usually considered that the small quantity required for this purpose cannot be productive of mischief; but it is forgotten that the liquid is employed by ignorant cooks who apportion the dose of poison by conjecture, and assuming that the greatest precautions are taken, it appears to me that a flavor is at all times dearly purchased if it depends on even a small dose of poison. Mr. Streeter met with a case in which a child suffered from symptoms of poisoning owing to its having eaten tapioca flavored with the oil of almonds. (Med. Times and Gaz. Dec. 16, 1854, p. 625.) There is a liquid sold for flavoring confectionery, under the name of *Essence of Jargonelle Pear.* It is a noxious artificial compound, made by distilling oil of grain or fusel oil, with acetate of potash and sulphuric acid. In the Pharmaceutical Journal (Nov. 1851, p. 214) it is stated that a child who had on two occasions eaten confectionery flavored with essence of pear, became partially comatose with livid lips and a feeble pulse. Symptoms resembling those of poisoning, occasionally observed among children, may often be referred to the eating of confectionery, colored or flavored with various kinds of poison. Among the artificial fruit-essences is one named *Essence of Ribstone Pippin,* or "oil of apples." It is procured from a mixture of bichromate of potash, sulphuric acid, and amylic alcohol (Chemical Record, Jan. 17, 1852, p. 44); all substances of a noxious nature. Butyric ether dissolved in six parts of alcohol is used as *Essence of Pineapple.* This ether appears to constitute the flavor of the pineapple, melon, and strawberry. Impure glycerine (the sweet principle of soap) mixed with alcohol, produces, by fermentation, a similar essence.

CHAPTER XXXIX.

BITTER ALMOND WATER—LAUREL WATER—LAUREL OIL—SYMPTOMS AND EFFECTS—CASE OF DONELLAN—SEEDS YIELDING PRUSSIC ACID—RATAFIA—CYANIDE OF POTASSIUM—SYMPTOMS—APPEARANCES—LOCAL ACTION—ALLEGED EFFECTS OF THE VAPOR—FATAL DOSE—ANALYSIS—SULPHO- AND FERROCYANIDES OF POTASSIUM—CYANIDES OF IRON, MERCURY, AND SILVER.

BITTER ALMOND WATER.

THIS water is made by distilling one part of almond-cake with eight parts of water. It varies considerably in strength. The late Dr. Gregory has stated that it contains one per cent. of anhydrous prussic acid. Mr. Bell informed me that in a specimen which he analyzed, the proportion of acid was only 0·27 per cent. I have met with specimens containing less than this. The odor is no criterion of the strength, since the odor of prussic acid is concealed by that of the bitter almond, and the odor may exist in a specimen of the water which contains no prussic acid. Its strength is impaired by keeping; thus Zeller found that one ounce of the water, fresh made, yielded 5·12 grains of cyanide of silver;

but after one year, when merely corked in a bottle, the proportion yielded was only 4·62 grains. (Pharmaceutical Journal, February, 1846, 371.)

Symptoms and Effects.—This water is poisonous, and on one occasion the improper use of it led to a criminal trial (Reg. v. Cronin, Central Criminal Court, April, 1847). The accused was charged with the manslaughter of a female under the following circumstances. He had been in the habit of using a preparation which he called bitter almond water, made by mixing three drops of the essential oil with a pint of water—a harmless mixture in small doses. He wrote a prescription for the deceased, in which occurred the words, *Aquæ amygd. amar.* Six ounces of this were ordered, and the mixture contained besides a small dose of prussic acid. The chemist who prepared the mixture put into it six ounces of the liquid commonly known as "bitter almond water" (distilled from the cake). The deceased took a tablespoonful and a half. In three minutes she said, "Oh, how queer I feel!" She left the room and ran out towards the garden, where she fell, breathing hard and groaning. There was dilatation of the pupils with general relaxation of the limbs; but there were no convulsions. She died shortly afterwards. There was no doubt that the bitter almond water had caused her death. The viscera were generally healthy. There was no odor in the abdomen; but it was perceptible in the brain, the vessels of which were somewhat congested. Prussic acid was detected in the stomach. The accused was acquitted, as it was not considered that he was strictly responsible for the result. (Med. Gaz. xxxix, 388 and 695.) The quantity of anhydrous prussic acid which the deceased took was equivalent to 0·94 grains; thus bearing out, in a most striking degree, the assumed fatal dose of this poison (p. 583), and proving that dilution with water does not prevent or materially retard its action on the body.

This water is not commonly employed medicinally in England, as its effects are very uncertain. Eighteen drops have been known to produce giddiness, dimness of sight, and a tendency to sleep. Twenty-two drops caused convulsions and vomiting. MM. Duvignan and Parent, who tried these experiments on themselves, did not feel inclined to carry the dose further. A drachm of the water killed a moderate sized dog. (Paris, Med. Jur. 243.) In France, bitter almond water is used medicinally, in doses of from ten to forty drops.

Analysis.—The water is sometimes opaque, from a little oily matter diffused through it; but it may be rendered clear by alcohol. Some specimens will yield readily all the usual reactions with the liquid tests for prussic acid; but when the water has been long kept, it has often only the odor of the bitter almond, and contains no prussic acid. When it has been sufficiently strong to cause serious symptoms or death, there will be no difficulty in detecting prussic acid by the vapor or liquid tests. A few drops of the water warmed with the bihydrosulphate of ammonia, will give, on the addition of a persalt of iron, the red color indicative of sulphocyanic acid. The strength of bitter almond water is by no means proportioned to the quantity of bitter almonds used; but it varies according to the process employed for its production. The same weight of almonds has given two kinds of water,—one ounce of one giving as much as 5·35 grains of cyanide of silver, and one ounce of the other only 2·5. When the residuary almond-cake has been previously digested in spirit, the water obtained is always weaker. (Pharm. Jour. Feb. 1846, 371.) In this case the amygdaline is partly removed from it.

LAUREL WATER—LAUREL OIL.

Laurel water is a weak solution of prussic acid, containing only about one-fourth of a grain per cent. of the strong acid; but it is said to be more poisonous than this quantity of acid would indicate. (Pereira, vol. ii, pt. ii, p. 279.) The leaves gathered in wet and cold weather are said to yield more prussic acid than those gathered in hot and dry weather. (Zeller.) The old leaves yield much less oil and acid than the young and unexpanded leaves. In some specimens of the water which I procured by distilling the bruised tops and fine shoots of the laurel with water, the odor was powerful; but the proportion of prussic acid present was smaller than that above stated. Like bitter almond water, it is variable in strength. Specimens long kept and frequently exposed seldom contain any prussic acid, although the odor of bitter almonds may be strong. It is a limpid colorless liquid, producing, in large doses, the usual effects of poisoning by prussic acid.

By distillation with water, the leaves of the plant yield also an essential oil, *Cherry laurel oil*, resembling that of the bitter almond; but much weaker, as it contains on an average less than three per cent. of prussic acid. A fluid ounce of water will dissolve only 3·25 grains of the oil. Every part of the plant is poisonous, but especially the leaves, flowers, and kernels of the fruit; these, when bruised, yield prussic acid, but the pulp of the cherry is not poisonous. Articles of food are often flavored with the leaves, and accidents are said to have arisen from this practice. (Pharm. Journ. July, 1847, 13.) The late Dr. Paris states that several children were severely affected by partaking of some custard flavored with laurel leaves, and were ill for three days. A girl of six and a boy of five years of age fell into a profound sleep, out of which they could not be roused for ten hours. (Med. Jur. ii, 402.) The leaves are often employed with impunity; but the proportion of oil and prussic acid is liable to vary with the age of the leaf. Dr. Christison states that he has found ten times as much oil in the young as in the old leaves, when both were gathered in May and June. (Op. cit. 788.)

Symptoms and Effects.—About half a teaspoonful of a mixture, consisting of four-fifths cherry laurel water, was given by mistake to an infant eight months old. The child threw its head back, was convulsed, and died in a few seconds. The laurel water taken in this case is said to have been stronger than usual. The body was inspected twenty-four hours after death. Nothing was observed in the brain and spinal marrow; but the stomach contained two teaspoonfuls of a yellowish liquid without odor, and its mucous membrane was reddened towards the greater curvature. No trace of prussic acid was found in the contents; but the poison was easily detected in the liquid remaining in the phial. (Med. Gaz. Jan. 1843.)

The following case is remarkable chiefly from the circumstance of the symptoms coming on slowly. A man swallowed one morning an ounce and a half of laurel water. No symptoms appeared until three hours afterwards. There were then numbness of the hands and feet, drooping of the head, and involuntary evacuations. The limbs became cold, and he lost all power over them, although sensibility was retained. The pulse was small; there was perfect consciousness. He gradually became weaker and died the same evening. On inspection, the only remarkable appearance was, that the blood was viscid and of a dark colour. There was no odor of bitter almonds. (Canstatt, Jahresbericht, 1844, v, 289.) A man, æt. 60, swallowed two ounces of laurel water of the Prussian Pharmacopœia. He fell almost immediately, as if in a fainting fit. He vomited some food which he had taken shortly before. He was seen in an hour by Casper, who found him on a sofa in a half-sitting posture, with his head falling forwards. His face was pale and cold, and the skin generally was cold. The pulse was slow, soft, and irregular. The most striking symptom was a general paralysis of the nerves of motion. He gave no sign of consciousness. The features were occasionally distorted with convulsive movements. There was no power of swallowing; and in this state the man continued, in spite of treatment, for *five hours*, when he died—probably the longest duration of any case of poisoning by prussic acid yet recorded. The only appearances in the body were congestion of the brain and right side of the heart, with a dark and liquid state of the blood. There was a smell of bitter almonds throughout. (Casper, Ger. Med. i, 431.) The appearances are similar to those met with in death from diluted prussic acid. Similar *treatment* is required (p. 588).

Cases of poisoning by laurel water are not common, and generally arise from accident. It has however been used for the purposes of murder, and of this the trial of Captain Donellan, at the Warwick Lent Assizes, in 1781, is a remarkable illustration. The accused was charged with the murder of his brother-in-law, Sir T. Boughton, by causing to be administered to him two ounces of laurel water, which he had criminally substituted for an innocent purgative draught. Admitting that the laurel water had no greater strength than that above assigned to it, the deceased must have taken 2.4 grains of pure hydrocyanic acid, a quantity equal to *fifty drops* of Scheele's prussic acid. The draught was administered to the deceased by his mother, Lady Boughton. She perceived that it smelt strongly of *bitter almonds*—the only evidence of the probable nature of the poison; for the original draught, containing rhubarb, jalap, spirits of lavender, and nutmeg water, would have had no such smell. The following were the symptoms: "In about two minutes after swallowing the draught, the deceased appeared to struggle very much, as if to keep it down, and had 'a rattling and gurgling' at his stomach. In about ten minutes, he seemed inclined to doze; and in about five minutes afterwards he was found with his eyes fixed upwards, his teeth clenched, and froth running out of his mouth." He died in half an hour after swallowing the draught. An examination of the body was not made until eleven days after death. The appearances were ambiguous, and no poison was detected in the body.

In making every allowance for such coincidences, in the supervention of fatal disease at the time of taking medicine and food, as have elsewhere been pointed out (p. 111), I do not think that there is any reason to doubt that in this case the deceased died from poison, and that the prisoner was properly convicted. It has been urged that the medical evidence was of itself insufficient; and that, without the moral circumstances, the charge of poisoning could not have been made out. But it is impossible to divide evidence in this way; it is like separating two series of circumstances in presumptive evidence, either of which, taken singly, may be weak, but when taken together, become strong. Many convictions on medico-legal trials for murder by poisoning would not have occurred if the fact had rested on moral or on medical evidence *alone*. In Donellan's case, the medical evidence was strong, whether we regard the time of the occurrence of symptoms, their character, their rapid course, or the period within which death took place. To exclude all notion of these effects depending on a draught just before taken, and having the decided odor of a liquid known to be capable of producing such symptoms, an odor which the originally prescribed draught could not possibly have had,—and to refer them to a disease, unusual in so young a subject, and unlikely to have caused death so rapidly, or under the symptoms actually witnessed—is simply to create impunity for the cunning and skill often displayed in murder by poison. In spite of the suggestions of Sir Fitzroy Kelly at the trial of Tawell (p. 601), and of Mr. Sergeant Shee at the trial of William Palmer (May, 1856), *direct* evidence can rarely be obtained in such cases. The murderer, unless insane, does not proclaim to the world his intention to poison another, nor the nature of the poison used; nor does he administer it openly, or call in "experts" to witness the administration and record the symptoms! Every minute circumstance, therefore, requires the closest watching and analysis if we wish to prevent, by punishment, this most detestable crime. The insisting upon *direct* evidence, in these cases, is tantamount to asking for impunity for the educated and skilful, or the professional poisoner,—and inflicting the full penalty of the law only on the uneducated and unskilful assassin.

Analysis.—The odor of the water is sufficient to identify it, but this will not prove that it contains prussic acid. In order to obtain this proof, it must be submitted to analysis. The following is the result of an examination of a very weak specimen: Nitrate of silver produced no perceptible effect with one drachm of it when the liquids were mixed, nor could any Prussian blue be procured from a like quantity by the use of the iron test. One drop of hydrosulphate of ammonia, added to three drops of the water and heated, gave the clearest evidence of prussic acid by the production of the red sulphocyanate of iron, when the persulphate of iron was added to the evaporated residue. .From five to ten drops placed in a watch-glass, produced no film (by the vapor) on nitrate of silver after the lapse of twenty minutes: in the same period of time, one drop of hydrosulphate of ammonia absorbed the vapor, and left, on evaporation, a perceptible quantity of sulphocyanate. Prussic acid is easily detected by the sulphur vapor test, in the shoot of the laurel, or in the seed of the berry (not in the pulp) when bruised and mixed with *cold* water. The application of heat to the pulp expedites the chemical change. The hydrosulphate of ammonia, added to the watery liquid of the pulp and heated, produces the usual reaction of sulphocyanate of ammonia with a per-salt of iron.

As it is desirable to have a corroboration of the action of the sulphur test when the analyst is dealing with small quantities, the following process will be found to present a satisfactory method of employing the silver and sulphur tests with one and the same portion of vapor. Receive the vapor on a drop of solution of nitrate of silver as described at p. 590. Examine it for crystals by the microscope: if the white film is slowly formed these will be visible. Now add a drop of bihydrosulphate of ammonia and warm the liquid,—black sulphuret of silver will be separated and by slow evaporation a watery-looking residue will result. This may be tested by adding persulphate of iron to the liquid; or, clean bibulous paper dipped into it, will remove the clear liquid from the black sulphuret of silver, and when the paper is touched with a solution of colorless persulphate of iron, the red color of the sulphocyanate will appear. The strength of laurel water is so variable, that it admits of no safe comparison with prussic acid: each specimen will require a separate examination. An *aqua lauro-cerasi* is used in the Dublin and Edinburgh Pharmacopœias. The French Codex prescribes the dose of from ten to forty drops every two hours. (Pharm. Jour. Feb. 1846, 372.)

The distilled waters of the leaves of the *Acacia*, and of the fruit of the passion flower, contain prussic acid. The Acacia water has a strong smell of bitter almonds, and eight ounces of it, precipitated by nitrate of silver, yielded 4·15 grains of cyanide. The dried leaves gave no prussic acid on distillation. The leaves of the peach yield a water as strong as that of the laurel. The distilled water of the *leaves* of the *Sweet almond* contains prussic acid. Zeller found that one ounce gave 0·575 grains of cyanide of silver. The flower of the common lilac is also said to yield traces of this poison.

NOYAU—CHERRY RATAFIA—APPLE SEEDS—PEACH KERNELS.

These, and all other liqueurs having the smell of bitter almonds, are considered to be poisonous when taken in large doses. The quantity of prussic acid present in them is liable to vary; it may be separated by distillation at a gentle heat in a water-bath, and then tested. I have found that an ounce and a half of good noyau, having a strong odor and flavor, yielded when distilled to two-thirds, scarcely a trace of prussic acid either by the silver or iron test. It had been kept some years in a well-corked bottle. An equal quantity of cherry ratafia, similarly treated, gave no ponderable quantity of Prussian blue. There are other plants, the leaves and kernels of which yield prussic acid; these are, the Bird-cherry, the Peach, Nectarine, Damson, Mountain-ash, Apricot, and the seeds of apples and pears. A case is reported in the Journal de Chimie Medicale, 1853, p. 38, in which a child, æt. 2, suffered severely in consequence of having eaten ten or twelve kernels of the apricot. I have examined the seeds of oranges and figs, but have found none; nor could I obtain the slightest trace of prussic acid from the distillation of three hundred grains of the *sweet* almond. The quantity produced from the seeds of apples has been grossly exaggerated (p. 602). The seeds of two large apples (seventeen in number) are equivalent in weight to one bitter almond (ten grains): but in the apple seeds, the quantity of husk is so large that it would probably require the seeds of four apples to produce as much prussic acid as one bitter almond. Ten bitter almonds, weighing 100 grains, will yield by distillation 0·24 grains of anhydrous prussic acid. It would require the seeds of forty apples to produce this quantity. In the unbroken state they would yield none, and they would pass through the body unchanged. If boiled (as in cooked apples,) so that the albuminous portion is coagulated, they would yield none, whether broken or unbroken. I have elsewhere alluded to this alleged production of a fatal dose of prussic acid from one of the most common articles of food (Case of Tawell, ante, p. 602). To have accounted for the quantity of prussic acid found in the stomach in that case, it must have been assumed that the deceased had eaten 160 apples or the pips removed from them, and that the pips were in a proper state for distillation after they had been swallowed!

Fresh and dried cherries, as well as the kernels and stones, yield prussic acid by distillation. The quantity yielded by the pulp of the cherry is very small, amounting to mere traces, but it is much greater in the stones and kernels. From sixteen ounces of cherry-stone water, Geiseler obtained 1·9 grains of cyanide of silver; and from cherry-kernel water, the kernels being to the water as 1 : 8 by weight, the cyanide of silver obtained from sixteen ounces was equal to 2·36 grains. Twelve ounces of the *kernels* yielded 7· grains of hydrocyanic acid: but the proportion of prussic acid yielded by the same weight of cherry-*stones*, according to Geiseler, was not more than 2·3 grains. (Pharm. Jour. Feb. 1846, 372.) These kernels, or bitter almonds bruised, are much employed for the purpose of giving a nutty flavor to alcoholic liquids. (British port.) It is not often that they are used in such quantity as to occasion accidents: but the following case, the details of which are somewhat imperfectly given, will show that the eating of a large quantity of the kernels may operate fatally.

A girl, æt. 5 years, ate a large quantity of the kernels of sweet cherries (prunus avium). Her brother (a few years older than herself) also ate some. After the lapse of a few hours, symptoms of poisoning appeared. When seen by a medical man on the next day, the girl was in such a stupor, that she could not be roused. The eyes were closed, pupils considerably dilated, the skin moist and hot, respiration exceedingly hurried, pulse small and quick, and the evacuations were discharged involuntarily; the child was very restless. An effervescing mixture was ordered internally, and cold fomentations were applied to the head; after a few hours, vomiting of a greenish substance ensued, and this was followed by retching, which continued until death; the body was spasmodically drawn backwards. The illness lasted forty hours. On an examination, the stomach was found intensely reddened; the intestines were strictured and invaginated (intussception), but there was not any inflammation. The liver, spleen, and large vessels, contained a black tar-like blood. The boy, who had eaten fewer cherry-kernels, was likewise ill, but recovered in the course of a month. An eruption, analogous to nettle-rash, showed itself on the arms of both children; they were both perfectly well (according to the statement of the mother) before eating the cherry-kernels, and no other cause for the attack could be assigned. The kernel of the prunus avium (cerasus nigra) contains amygdaline, and produces prussic acid as well as essential oil in the stomach. (Philadelphia Med. Exam. July, 1845, 490.)

A singular case of poisoning by *peach-kernels* was communicated to me by Mr. Hicks. A medical man swallowed half an ounce of liquid made by digesting gin on a large quantity of peach-kernels. He became giddy and had violent constriction of the throat and dimness of sight. He vomited and recovered. The bottle was brought to me by Mr. Hicks,—a few drops of the liquid contained in it, yielded only a faint trace of Prussian blue. The kernels weighed 124 grains; they were large and the skins entire. All the amygdaline must have been extracted, for on bruising the kernels with water and distilling them, not a particle of the poison could be procured. The amygdaline may have been converted to prussic acid in the body. (See p. 87.)

IATROPHA MANIHOT—CASSAVA.

The root of one variety of this West Indian plant, known under the name of Bitter Cassava, contains in its juice prussic acid. It is, therefore, when recently expressed, highly poisonous, inducing coma, convulsions, and death. Prussian blue may be obtained from the fresh juice by the iron test for prussic acid. The vegetable principles of the plant, evaporated to dryness, form what is called *Cassava-cake*, which is not only inert, by reason of the poison being volatilized, but highly nutritious. The starch obtained from this root is well known under the name of *Tapioca*. Neither cassava nor tapioca yields any trace of prussic acid.

CYANIDE OF POTASSIUM.

Prussic acid is as fatal to animal life when combined with alkaline bases as when it is free. Thus the same quantity of diluted prussic acid will kill a dog whether it be given in a pure state or combined with ammonia or potash. (Orfila, ii, 292.) Hence, ammonia cannot be regarded as a chemical antidote in cases of poisoning by prussic acid: it acts merely as a stimulant. Alkalies have not even the power of fixing the acid. In Reg. v. Cronin (C. C. C. 1847) the prisoner had prescribed aromatic spirits of ammonia with prussic acid, and the question was, whether the poison would become thereby in any degree disarmed of its virulence. The answer was in the negative, as the rapid death of the female proved. When mixed with an alkali, such as ammonia, prussic acid is liable to undergo speedy decomposition. It becomes yellow, then brown, and finally, almost black, a thick black sediment being formed in it. This change is, however, only partial: in one specimen thus altered, I found a large quantity of free prussic acid after two years. The change is not observed to take place when the prussic acid bears a small proportion to the alkali, or *vice versa;* nor does it so readily occur when the prussic acid is diluted.

The only saline compound of prussic acid which is of any practical interest as a poison, is the Cyanide of Potassium.

Symptoms.—This salt has a bitter taste, producing first a sense of coldness on the tongue, followed by a feeling of constriction, and burning heat, in the throat. It is one of the most formidable poisons known to chemists. It has led to the destruction of life in many instances within the last few years, chiefly owing to its having been administered by mistake for other medicinal preparations. The symptoms which it produces are similar to those occasioned by prussic acid: insensibility, spasmodic respiration, convulsions, with tetanic stiffness of the jaws and body. They appear in a few seconds or minutes, and run through their course with great rapidity. Orfila found that four grains and a half dissolved in water, and injected into the throat of a dog, produced well-marked symptoms in ten seconds; and there was convulsive respiration, followed by apparent death in five minutes. Vomiting of a liquid smelling strongly of prussic acid took place, and the animal died in eleven minutes. M. Bonjean gave to a rabbit 8-10ths of a grain of cyanide of potassium dissolved in a small quantity of water. The animal had a violent convulsive fit, and died before it could completely swallow the poison. This gentleman found that animals to which the poison was given invariably had convulsions, tetanic spasms, and abdominal respiration. When administered with some kinds of white wine containing iron, he found that it gave a bluish tinge to the mucous membrane of the alimentary canal. (Faits Chimiques rel. à l'Emp. par l'Acide Prussique, 1843, p. 30.) Bischoff states that a titmouse was killed in half a minute by 1-18th of a grain of the cyanide placed on the tongue, and that a guinea-pig died in violent convulsions in a few minutes from a grain and a half placed on the tongue. (Canstatt's Jahresbericht, 1844, B. v, p. 292.)

The Cyanide of Potassium is much used on the continent as a medicine, and some years since it occasioned the death of a person at St. Malo, under the following circumstances. A physician prescribed for the deceased rather more than one drachm of the cyanide in two ounces and a half of orange-flower water and syrup; and of this mixture three spoonfuls were to be taken daily. It seems that a tablespoonful was taken for the first dose, and the patient died in three-quarters of an hour. None of the poison was found in the stomach; but a portion of the mixture from which the first dose had been taken was examined, and found to contain the cyanide in solution. A criminal procedure was instituted against the physician, and he was fined and imprisoned. MM. Malaguti, Sarzeau, and Guyot, who gave evidence on the occasion, stated that they found no trace of the poison in the body,—that the cyanide was pure, and only one tablespoonful was missing from the bottle. They further stated, that a dog was killed in a few minutes after taking less than *three grains* of the cyanide in solution, and that the largest medicinal (?) dose to a human being was five-sixths of a grain. (Lancet, Jan. 1843.) The mixture in this case, contained about three grains of the cyanide in one drachm: therefore had teaspoonfuls been taken by the deceased, the quantity would have been quite sufficient to destroy life. The medicine had evidently been prescribed by a person totally ignorant of its poisonous properties. (Ann. d'Hyg. 1843, i, 413.) Another case occurred at Breslau, in January, 1842, in which a man, æt. 30, died in a quarter of an hour under all the symptoms of poisoning by prussic acid, after taking a dose of a mixture which had been prescribed for him by his medical attendant. (Henke's Zeitschrift der S. A. 1843, p. 7; and Ann. d'Hyg. 1843, i, 404.) Fifteen grains of "kali hydrocyanicum" in a dose, were prescribed by a physician for his patient; he intended to order the ferrocyanide of potassium, but instead of this salt, cyanide of potassium was sent: the patient took the poisonous draught. On inspection, there was no particular odor: but the poison was detected in the contents of the large intestines.

In January, 1844, a similar accident occurred in Germany, by which the patient was killed, and the physician had a narrow escape of his life. Two drachms of "kali hydrocyanicum" were ordered in a prescription, with two drachms of sugar dissolved in two ounces of camomile-water; a dessert-spoonful to be taken every quarter of an hour. Cyanide of potassium was dispensed instead of the ferrocyanide—the salt intended! The patient, an adult, took a dose (about 100 drops), and the operation of the poison was manifested during the act of swallowing. There was a tendency to vomit, and an immediate loss of consciousness: death took place in an hour. The quantity of cyanide here taken was not less than from thirteen to fifteen grains, equivalent to more than five grains of anhydrous, or 100 drops of Scheele's prussic acid! The physician who prescribed the medicine was sent for while the patient was still suffering from its effects: and in order to show that he had prescribed an innocent mixture, he put about a teaspoonful of it into his mouth, and swallowed three-fourths. The remainder he spat out, as it gave him an astringent or constricting sensation in his throat, like that caused by alum or green vitriol. He immediately felt severe pain in the back of the head, there was inability to stand, indistinct vision, nausea, a rushing sound in the ears, loss of consciousness, and, without complaining of any well-defined pain, he felt that he had lost the power to make a deep inspiration. The loss of sense was as rapid as in ordinary syncope. When an effort was made to swallow some milk, there was a strong feeling of choking, followed by copious vomiting. For more than an hour he could not stand upright. Giddiness, weight in the head, and constriction in the throat, continued for many hours. He passed a restless night; but the next day, with the exception of suffering from a general relaxation and weakness, he had recovered, and was enabled to assist at the examination of the body of his unfortunate patient! (Casper's Wochenschrift, Oct. 1845, p. 657.) This case shows the necessity of great caution in prescribers who make themselves the subject of experiment in swallowing what they suppose to be their own medicine (see p. 115).

Insensibility is not always an immediate symptom. A woman, who at the time was under medical treatment, took by mistake a teaspoonful of a solution of cyanide of potassium, this quantity containing about seven grains of the salt. Immediately after taking it she complained of a severe burning pain in the stomach, and a feeling as if the bowels were about to act. She went to the water-closet, and her strength left her. She was removed to bed, and speedily became unconscious. It was found impossible to introduce anything into the stomach. She died in less than an hour. There was no convulsion before death, but a sudden convulsive action of the body took place after the heart had ceased to beat. The appearance of the body was so natural, even on the day following death, that some of her friends supposed there might still be life. (Boston Medical and Surgical Journal, Dec. 11, 1856, and Brit. and For. Med. Rev. 1857, vol. xix, p. 498.)

In June, 1856, a woman swallowed an ounce and a half of a solution of cyanide of potassium, used for photographic purposes. The quantity taken amounted to five grains. In two minutes she became unconscious, the whole of the body was slightly convulsed, and the pupils of the eyes were dilated. She foamed at the mouth, the pulse was small and feeble, and there was spasmodic closure of the jaws. Nevertheless, as she had lost some teeth, there was sufficient space for the introduction of the tube of the stomach-pump, within five minutes after she had taken the poison. She died in twenty minutes. In the Registrar-General's Report for Oct. 3, 1857, three deaths are stated to have occurred among the families of soldiers, two cases of suicide and one of accident. It appears that they employed it for cleaning lace. (For other cases of its fatal action, see Med. Times and Gaz. Oct. 12, 1850, p. 390; Nov. 9, 1850, p. 482; and July 12, 1851, p. 41.)

Appearances.—In the bodies of animals examined recently after death, an odor of prussic acid has been perceived. There was an ecchymosed condition of the mucous membrane of the stomach, and congestion of the vessels of the brain with dark-colored blood (Ann. d'Hyg. 1843, i, 409).

In the case quoted from Casper (p. 628) the inspection of the body was made two days after death: there was no remarkable odor: the muscles were stiff and rigid; the face, and fore part of the trunk, pale; the back part livid, except those portions which had sustained pressure. The fingers and toes were convulsively bent inwards, the nails blue, eyelids half-closed, lips pale, the sinuses and cerebral vessels filled with bluish-red (blaurothem) blood. On making a section of the cerebellum and spinal marrow, bloody points were observed. There was infiltration of the lungs posteriorly, and on cutting into them, a strong odor of bitter almonds was perceived. A yellowish mucus was found in the stomach, which yielded on analysis cyanide of potassium. The mucous membrane was reddened near the pylorus. The poison was not detected in any part of the body except the contents of the stomach and intestines. (Casper's Wochenschrift, Oct. 4, 1845, 657.)

In Nov. 1851, a girl, æt. 18, was brought to Guy's Hospital. Half an hour before her admission she was seen to swallow a solution of cyanide of potassium. She vomited once. It is stated that she was alive when put into the cab, but when taken out at the hospital seven minutes afterwards, she was completely insensible, pulseless at the wrist, and to all appearances dead. Artificial respiration was at once resorted to, ammonia applied to the nostrils, warmth to the extremities, and cold affusion to the spine; but all to no effect, and with the exception of an alteration in the pupils observed during the first few minutes of her admission, she evinced no evidence of vitality. The body was inspected on the following day. The stomach contained a large quantity of half-digested food: its mucous membrane was of a pink color and deeply injected, especially in patches. The bronchial tubes contained a considerable quantity of a white substance, which on examination proved to be chalk. Every other organ was healthy, and there was no appearance of corrosion about the mouth. The contents of the stomach were of a pale straw color, semi-fluid, and had a decided bitter almond odor. Prussic acid was detected in them.

Local action.—Cyanide of Potassium possesses a *local* action. A patient was directed to use as an injection a solution of rather less than *five grains* (4·6 gr.) of the cyanide dissolved in six ounces and a half of water. He was seized soon afterwards with convulsions, palpitation, slow and difficult respiration, coldness of the skin, dilatation of the pupils, and fixedness of the eyes. He died in an hour. (Ann. d'Hyg. 1843, i, 412.) It appears that thirty-six hours previously, he had used a similar injection without injury, but it is probable, that the cyanide then employed was not pure.

Dr. Chanet has directed attention to the local action of this poison on the hands of the workmen engaged in the arts of electro-gilding and silvering. The hands of these men are almost always covered with ulcers. The skin about the joints is fissured, and an oozing of blood often takes place. The nail with its root participates in the inflammation. The workmen informed him that, on dipping their arms into the bath for a few seconds, the whole of the skin became reddened. The ulceration of the soft parts continued even to the bone, producing great pain and broken rest. (Gaz. des Hôp. 24 Juil. 1847, 374.) The strong alkalinity of the solution would explain some of these effects, for the solution readily dissolves the cuticle, and exposes the true skin. The excoriations produced may, however, lead to the absorption of the poison, and to all the effects of chronic poisoning by prussic acid. The extensive use of the cyanide in photography has led to other accidents. The salt has been used either in a strong solution or in a solid form for the purpose of removing stains on the fingers. If there should be any cut or abrasion of the skin, the poison may be absorbed and produce serious symptoms. Dr. Atherstone is said to have suffered from all the symptoms of poisoning by prussic acid as a result of this local use of the cyanide; and other cases of severe local inflammation are reported. There can be no doubt that the use of this salt for such purposes is a dangerous practice.

It has been supposed that the cyanide of potassium might exist in the state of *vapor*, and destroy life by its accidental introduction into the lungs. When the cyanide is exposed to a damp atmosphere, or is acted upon by acids, hydrocyanic acid freely escapes, and the respiration of this vapor may produce injurious or even fatal effects. It does not appear probable, however, that the cyanide should itself be respired in the state of vapor. In December, 1853, an inquest was held at Elsecar by Mr. Badger, under the following remarkable circumstances: Three members of a family named Sadler, and a lodger, went to bed in their usual health at about ten o'clock, sleeping in different bedrooms. At seven on the following morning they were all found dead. The house, in which this accident occurred, abutted on one of the blast-furnaces of the Elsecar Iron-works; and it was obvious that some noxious vapors from the furnace must have escaped into the rooms through a crack in the house-wall. It was considered by a gentleman who examined the premises, that the noxious agent in this instance was the cyanide of potassium in vapor: but as this salt is not volatile under a white heat, —is only evolved in the lower part of iron furnaces, and cannot be carried far without condensation, it is difficult to conceive how it could exist and spread itself in the form of respirable vapor through the air of the apartments in which the deceased were sleeping. The more probable explanation, as it appears to me, is, that carbonic oxide or nitrogen from deoxidized air was the agent of destruction in this instance, supposing that no carbonic acid was formed by the combustion of the carbonic oxide. It is inconceivable that a substance which remains fixed at a heat of 1000° and upwards, should be diffused at a distance in the form of vapor through air at a common temperature; and nothing short of its detection in and upon the bodies of the deceased could have warranted the admission, that the respiration of this substance in vapor was really the cause of death.

Fatal dose and period of death.—Two grains and a half (2·44 gr.) of the pure salt are equivalent to one grain of anhydrous prussic, or fifty minims of the London pharmacopœial acid. Hence the cyanide may be regarded as a solid compound of hydrocyanic acid containing of this poison in its most concentrated form, no less than 39·3 per cent. by weight! A dose of from three to five grains of the pure salt may, therefore, easily prove fatal. From a case just related, it would appear that a dose of less than *five grains* has actually destroyed life, and in another case *five grains* proved fatal. Death has taken place in a quarter of an hour, but it may prove even more rapidly fatal.

The energy of cyanide of potassium as a poison depends, in some measure, on its mode of preparation. Some specimens are so impure as to contain a large quantity of carbonate of potash, from which the cyanide may be separated by its ready solubility in weak alcohol. (See Ann. d'Hyg. 1843, p. 404, in which this subject is fully investigated by Orfila.)

The cyanide is not used medicinally in England. The *medicinal dose* is estimated as from one-eighth to one-fourth of a grain; but, as the salt is of uncertain composition, it is a most dangerous substance to employ. From its great solvent powers on the metals, it is extensively employed in the arts of electro-gilding and plating, as well as in photography. The solution is improperly kept exposed, and is constantly evolving hydrocyanic acid in vapor.

Treatment.—The symptoms occur with such rapidity and violence, that there is scarcely time to employ treatment. The administration of a weak solution of green sulphate of iron would have the effect of decomposing the poison, and converting it to Prussian blue. Cold affusion and the other remedies used in poisoning by prussic acid should be also applied.

CHEMICAL ANALYSIS.

When pure, the cyanide appears as a white crystallized salt, or as a white-looking mass. It has an acrid alkaline bitter taste; it is without any odor, until put into water, or until air and moisture have had free access to it; it then has the well-marked odor of prussic acid. It is deliquescent, and very soluble in water: the solution, when pure, is colorless, and has a strong alkaline reaction, a soapy feel, and a powerful odor of prussic acid. It is not very soluble in pure and strong alcohol. 1. It is decomposed by all acids, and prussic acid is set free. 2. The potash is precipitated by tartaric acid and chloride of platina. 3. It gives a white precipitate with nitrate of silver, which, when dried and heated, possesses all the properties of cyanide of silver (ante, p. 590, and 623). This precipitate is easily redissolved by a slight excess of the solution of cyanide of potassium. 4. If a solution of green sulphate of iron be added to it, and, afterwards, diluted muriatic acid, Prussian blue will result, indicating thereby the true nature of the salt. 5. A single grain of this salt moistened with water in a watch-glass, gives a well-marked reaction, by its vapor, with the silver and sulphur tests. Should this experiment fail, a drop of the bihydrosulphate of ammonia may be heated with the cyanide,—the liquid acidulated with hydrochloric acid and a solution of persulphate of iron added. The red color of the sulphocyanate of iron is immediately brought out.

Organic liquids.—The liquid may have the odor of prussic acid. A small portion should be filtered for preliminary testing. If the cyanide be present, the addition of a solution of green sulphate of iron and hydrochloric acid will produce Prussian blue before distillation. By distilling the organic liquid with sulphuric acid, prussic acid is obtained in the receiver, and sulphate of potash may be procured by incinerating the residue left in the retort. Advantage may be taken of the insolubility of this salt in pure alcohol, to separate from it some organic principles. M. Bonjean found in one experiment that cyanide of potassium was entirely lost as a result of decomposition in the dead body of an animal. After forty days he was unable to detect it, either by the odor or by tests, in the stomach of a rabbit, which had been killed by 8-10ths of a grain, and into which a like quantity had been introduced soon after death. (Op. cit. p. 33; see also p. 627.) It is probable that it may be changed during putrefaction into sulphocyanides of potassium and ammonium. It should then be sought for by digesting the viscera in a mixture of alcohol and water.

SULPHO- AND FERROCYANIDE OF POTASSIUM.

The sulpho- and ferrocyanic acids, whether free or combined with alkalies, are said not to be poisonous; but further experiments are required to determine to what extent they may be noxious to man. A singular case, in which *Sulphocyanic acid* was alleged to have been the cause of death, will be found reported in the Brit. and For. Med. Rev. July, 1839. A man wishing to destroy himself, swallowed a liquid which he had obtained by distilling strong sulphuric acid with ferrocyanide of potassium. He was found dead in his room, and twenty-four hours afterwards the body was examined. The stomach was not inflamed, but part of its mucous surface was softened, and of a brownish-black color. There was no odor of prussic acid. Some doubt being entertained as to what the products of such a distillation might be, experiments were performed; but the results obtained by the different experimentalists did not agree. In repeating the distillation, I have found that prussic acid in large, and sulphocyanic acid in small quantity, were procured; and it is highly probable that death was really caused by prussic acid, which may have been the case, although no odor was perceptible. The blackened state of the stomach was probaby due to some strong sulphuric acid being mixed with it.

Sulphocyanic acid and *Sulphocyanide of potassium* have been found, in moderately large doses, to cause the death of animals. Bernard, who has experimented on this subject, states that the sulphocyanide produces direct paralysis of the muscular system, and arrests the action of the heart: but this was observed only where the poison had been introduced directly into the circulation—a case which is not likely to present itself in medical jurisprudence. A solution of it injected into the stomach or under the skin of a rabbit, produced no symptom of poisoning. (Leçons sur les Substances Toxiques, 1857, pp. 351, 356, 386.) Bernard ranks this as a blood-poison of great power. The salt is generally present in small quantity in saliva: hence it must be formed and secreted by the blood. The excess of it in the saliva of dogs has been supposed, but on no satisfactory grounds, to account for the occurrence of rabies in these animals. The salt, unlike the ferro- and ferricyanides of potassium, is a remarkable deoxidizer, and its noxious operation in the blood may depend upon its removing oxygen from that fluid. With respect to the *ferrocyanide* and the ferricyanide of potassium, Bischoff found that five grains produced tremors in a small rabbit. These passed off; the animal ate its food readily, but died in five days. On a charge of poisoning with the ferricyanide of potassium, which occurred in Germany, the medical witnesses were asked whether it was a poison. They could not answer the question, but said it would undergo a decomposition in the stomach which would render it inert! In this instance there was no proof that the salt had even been swallowed; and the sudden death of the woman appeared to be due to hydrothorax. (Canstatt's Jahresbericht, 1844, B. v, s. 291.) Any acids in the stomach would tend to decompose it and set free prussic acid; but this change has but little tendency to take place at the temperature of the body (98°). Further experiments are required to determine the properties of this compound. According to Schubarth, it is not poisonous to man or animals in drachm doses. It was formerly supposed to contain prussic acid, and that it was therefore highly deleterious. It is now known, however, that prussic acid is a product resulting from a reaction of its elements, and that it has no independent existence in the salt.

Analysis.—*Sulphocyanide of potassium* is a white crystallizable salt, very soluble in water, and forming with it a colorless neutral solution. The tests which may be employed are—1. *Persulphate of iron.* If persulphate of iron be added to a solution of this salt, even when in small proportion, it immediately produces a deep blood-red color. The color is destroyed, and a milky-white precipitate is thrown down on the addition of a solution of corrosive sublimate. 2. *Iodic acid.* When added to the solution, iodine is set free, indicated by the blue color pro-

CHAPTER XL.

POISONING BY ALCOHOL—SYMPTOMS OF ACUTE AND CHRONIC POISONING—APPEARANCES—FATAL DOSE—TREATMENT—ANALYSIS—ETHER-VAPOR—SYMPTOMS AND EFFECTS—APPEARANCES—LIQUID ETHER—ANALYSIS—CHLOROFORM VAPOR—SYMPTOMS—APPEARANCES—DEATH FROM LIQUID CHLOROFORM—TREATMENT—ANALYSIS—POISONING BY CAMPHOR—SYMPTOMS AND APPEARANCES.

ALCOHOL.

THE only form of poisoning by alcohol, which a medical jurist has to encounter, is that which arises from the taking of large quantities of spirituous liquors—such as gin, whiskey, rum, or brandy. The two last-mentioned compounds contain about fifty-three per cent. by measure of alcohol, while gin and whiskey are rather stronger,—gin containing as much as fifty-seven per cent.

Symptoms.—A large quantity of spirit has been known to destroy life immediately, although such a case is rare. Orfila mentions an instance in which a man died immediately from the effects of a large dose of brandy. (Op. cit. ii, 528.) In general, the symptoms come on in the course of a few minutes. There is confusion of thought, with inability to stand or walk, a tottering gait and giddiness, followed by stupor and coma. Should the person recover from this stage, vomiting and sickness supervene. This form of poisoning presents some singular anomalies: thus the insensibility may not come on until after a certain period, and then suddenly. Dr. Christison met with an instance in which a person fell suddenly into a deep stupor, some time after he had swallowed sixteen ounces of whiskey—there were none of the usual premonitory symptoms. In another instance, a person may apparently recover from the first effects,—then suddenly become insensible, and die convulsed. Convulsions are, however, by no means a necessary attendant on poisoning by alcohol. Orfila makes their absence a ground of distinction between poisoning by alcohol and opium (Op. cit. ii, 530),—and Dr. Ogston only noticed their occurrence twice out of many cases, and the subjects in these two instances were young. There is a ghastly or vacant expression on the features, which are sometimes suffused and bloated, the lips are livid, and the pupils are dilated and fixed. If they possess the power of contracting under the influence of light, this is a favorable sign. (See case in Lancet, Jan. 27th, 1855, p. 89.) The conjunctivæ or whites of the eyes are generally much suffused. In poisoning by alcohol the supervention of symptoms is not commonly so rapid as to prevent a person from performing locomotion or certain acts of volition. The more concentrated the alcohol, the more rapidly are the symptoms induced, and they are then more severe in their character. Diluted alcohol generally produces a stage of excitement before stupor, while in the action of concentrated alcohol there may be profound coma in a few minutes.

Alcohol may act as a poison by its *vapor*. If the concentrated vapor be respired, it will produce the usual effects of intoxication. It is generally known that persons who have been for the first time employed in bottling spirits, are easily intoxicated by the alcoholic vapor. There is a case on record in which a child two years of age was thrown into an apoplectic stupor by the alcoholic vapor of eau de Cologne. In this manner a child might be destroyed, and no trace of the poison be found in the stomach.

A man, æt. 27, was admitted into Guy's Hospital in Jan. 1857. He had drunk a large quantity of brandy about ten hours previously. He was observed to stagger immediately, and in spite of the use of the stomach-pump he continued in an insensible state. When brought to the hospital, he was in a state of collapse,—the skin was cold,—the pulse scarcely perceptible, and the pupils were greatly dilated. The contents of the stomach were drawn off by the pump and reserved for analysis: they had no odor of brandy. After a few hours he rallied slightly, and asked for something to drink; but he gradually sank again into a comatose condition,—the pupils remained dilated, the breathing was slow, and the face congested. He died twenty-two hours after taking the spirit.

A girl, æt. 7, was admitted into the Westminster Hospital in December, 1846. The child had swallowed eight ounces of undiluted rum during the absence of her parents from the room. Five minutes afterwards they found her lying on the floor insensible. On admission, twenty minutes after taking the rum, she was perfectly comatose,—the face was pale and bedewed with perspiration, the pupils were much contracted, the limbs were relaxed, the pulse was quick and jerking, the skin cool and moist, the breathing was scarcely perceptible. The stomach-pump was used, and the tepid water employed came away with a strong spirituous odor. The child was temporarily roused from the comatose state by cold affusion applied by means of a watering-pot. She continued in a state of insensibility for six hours, and the pupils were then much dilated. The comatose condition ceased in about eight hours after she had taken the alcohol: she was then sensible, and answered questions readily. (Medical Times, January 16, 1847, 313.) The recovery here must be ascribed to the early and energetic treatment. In some respects, it will be observed, this case resembled one of poisoning by opium. A boy, æt. 8, was found insensible about half an hour after he had swallowed about half a pint of gin. The liquid, drawn from the stomach seven hours afterwards, had no odor of gin: nor was the odor perceptible in the breath. He was insensible and motionless, his limbs were relaxed and powerless, his face pale, and his skin cold. The pulse was quick, small, and feeble. He died without rallying or recovering his consciousness, sixty-seven hours after taking the poison.

One of the remarkable features of poisoning by alcohol, seen in two of the above cases, is that a *remission* of the symptoms is by no means unfrequent, and that death sometimes takes place suddenly after some hours or days, when a person appears to have recovered entirely from the effects. A man, æt. 26, drank after dinner, in the course of a few hours, a pint of whiskey, and during the evening of the same day sixteen ounces of raw rum, one-half of which he drank at once, and the other half in three minutes. In five minutes he was found fast asleep, snoring, his mouth open, and saliva flowing from it; he was quite insensible, and had fallen backwards in his chair; his face was pale and cold, the skin cold; the breathing stertorous; there was no pulse; the eyes were half open, and the pupils unequally dilated and insensible to light. The stomach-pump was used, and a quantity of spirituous liquid drawn off. The following day he was rational, but remembered nothing of what had passed; he was feverish, the pulse rapid, and breathing quick. He complained of pain at the pit of the stomach, and vomited all that he took. On the third day he was suddenly attacked with great difficulty of breathing, and died in about an hour. The cause of death in most cases of acute alcoholic poison may be traced to congestion of the brain or lungs, or both.

Chronic Poisoning.—When alcohol has been taken for a long period in the shape of intoxicating drinks, the person suffers from a series of diseases, the characters of which are well marked. The usual effects are irritation of the stomach and intestines, nausea, vomiting, purging, jaundice, cerebral congestion, scirrhus of the stomach, dropsy, diabetes, paralysis, *delirium tremens*, and insanity. Such persons are subject to sudden death by coma. After death morbid changes are discovered in various organs; and the liver is especially affected. This organ is commonly enlarged, and of a lighter color than natural: it is called the nutmeg, or the drunkard's liver. It is not unusual to find the kidneys in a state of granular degeneration. In the body of a drunkard examined twenty-four hours after death, Dr. Albers found the convolutions of the brain of a pale yellow hue; the lateral ventricle full of air; and the cerebral substance unusually tough. Under the microscope there appeared in the gray cortical substance large oil-globules and fatty granular matter, both upon the upper surface and at the base; the fibrous structure was faintly marked; the cerebral ganglia were small; there were many oil-globules infiltrated into the white substance; the brain smelt like must, and half a drachm of rectified spirit was distilled from it. (Med. Times and Gaz. July 16, 1853, p. 72.) Of all the common consequences of the abuse of alcoholic liquids, *delirium tremens* is by far the most frequent. Although a result of chronic poisoning, a state analogous to it has been known to supervene rapidly, as in the following case: A boy, æt. 5, swallowed a large quantity of brandy. Vomiting speedily followed, and he passed a restless night. In the morning it was observed that he had trembling of the hands, and that he could not hold a cup steadily. Convulsions with cramps ensued. The pulse was slow; the look timid, the pupil dilated, and the face pale. Delirium supervened, and there was difficulty of passing urine, with great thirst. Under treatment, the symptoms abated, but there was a return of the trembling towards evening. An opiate was given, and the symptoms disappeared. (Gaz. des. Hôpitaux, and Med. Gaz. xxxviii, 554.) Delirium tremens is sometimes observed when, after long abuse, alcoholic liquids are suddenly discontinued: in these cases it is the result of the withdrawal of the stimulus, hence the symptoms are often mitigated when the use of alcohol is resumed. Something analogous to this is observed in chronic poisoning by opium.

were healthy; the gastric veins distended, and the liver congested. On opening the stomach there was an odor of ardent spirits. The mucous membrane from about half way up the gullet, and for eighteen inches along the intestines, was found highly injected. The mucous membrane of the stomach was of a deep crimson color. There was general congestion of the vessels of the brain and membranes. The man had obviously died from apoplexy, brought on by the dose of alcohol. (Lond. and Ed. Mon. Jour. June, 1844.)

Casper examined, on the fourth day after death, the body of a man who had died from excessive drinking. Cadaveric rigidity was well marked, and there was an absence of putrefaction. The skin was in a state of contraction (cutis anserina). The bloodvessels of the membranes of the brain were congested, and on the right hemisphere there was an extravasation of fluid blood. The great vessels of the chest were filled with dark liquid blood,—the lungs were normal; the heart was empty. There was an odor of alcohol in the head and chest. In another case, after seven days, there was scarcely any sign of putrefaction. There was congestion of the brain;—the blood was dark and fluid, and the cavities of the heart contained only a small quantity. The odor of brandy was perceptible in the head and chest. In a third, on the ninth day after death, the body was comparatively fresh: the inspection was made on the eleventh day. The membranes of the brain were congested: the lungs were œdematous. The right cavities of the heart were strongly distended with dark fluid blood: the left cavities were almost empty. The urinary bladder was distended with urine, apparently from a want of power to pass it. There was no odor of alcohol in any part. (Ger. Med. i, 453.)

Dr. Voltolina has described a cinnabar-red color of the pulmonary and aortic valves, among the appearances of death from alcohol. The color cannot be wiped out, and gives to the valves a very beautiful appearance. A remarkable quantity of dark thin blood was found also in the vena cava ascendens and the subclavian vein. Dr. Santlus met with a similar appearance of the valves in 1856. It remains to be proved whether this appearance is characteristic of alcoholic poison alone, or is found in other affections inducing fluidity of blood. (Berlin Med. Zeitung, 1857, No. 12; Med. Times and Gaz. May 1, 1858, p. 457.)

In a dose of poisoning by rum, recorded by Dr. Percy, there was great rigidity of the muscles, with a puckered state of the skin (cutis anserina) two days after death. The feet and hands were livid. The lungs were gorged with dark-colored blood. The air-tubes contained a frothy mucus: and there was some effusion under the mucous membrane of the glottis. The right cavities of the heart, with the large venous trunks, were distended with dark fluid blood. The left cavities and the aorta were empty. The stomach was reddened at the cardiac orifice, but otherwise healthy, as well as the intestines. The stomach contained a dark fluid, which smelt strongly of rum. The urinary bladder was distended. The membranes of the brain were gorged with dark fluid blood. The substance of the brain was firm, but presented many bloody points. It had an odor resembling that of rum. Dr. Percy obtained alcohol by distilling the substance of the brain. (Inquiry on Alcohol, p. 47.) In the case which occurred at Guy's Hospital (p. 636), there was lividity of the skin. The mucous membrane of the stomach was pale, excepting near the intestinal end, where there was a large red patch of capillary injection, such as would be produced by irritation. The intestines presented nothing unusual. The liver and other soft organs were of a dark color, as a result of congestion. The bladder was full of healthy urine. The brain in its substance and the membranes were congested with dark blood. There was a slight effusion of serum in the subarachnoid tissue: but there was no odor of spirit. I found alcohol in small quantity in the contents of the stomach, but there was none in the eight ounces of the substance of the brain which were submitted to distillation.

In the case of a man, æt. 26, who died on the third day (p. 636, ante) under symptoms of congestion of the lungs, the appearances after death were a red-colored patch on the mucous membrane of the stomach, and some redness of the intestines. There was effusion in the chest, but the heart as well as the brain and its membranes were healthy. (Med. Times, June 21, 1845, p. 219.) In several instances the brain has been found healthy, and the other appearances so slight as scarcely to call for notice. In the body of the boy, æt. 8, who died in sixty-seven hours (p. 636), the brain was healthy: there was slight effusion of a serous liquid, and the veins of the pia mater (inner membrane) were distended. The stomach was pale and free from any mark of inflammation.

Fatal dose.—The quantity of alcohol required to destroy life cannot be fixed. It must depend on the age and habits of the person. The smallest quantity known to have proved fatal was in the case of a boy, æt. 7, who swallowed two wine-glassfuls of brandy (between three and four ounces). Soon afterwards he was observed to stagger: he was sent to bed, and vomited violently. There was then a remission of the symptoms. He got up and sat by the fire: his head, face, and neck were very red, and he was in a profuse perspiration. Half an hour afterwards, he was found perfectly insensible, strongly convulsed, and the skin cold. He died in about thirty hours. The strength of the alcoholic liquid taken will materially influence a medical opinion in such cases. In a case in which I was consulted in March, 1857, a man drank two bottles of port wine (containing eleven ounces of alcohol) in less than two hours. He speedily became intoxicated and utterly helpless, and died, without rallying, from congestion of the brain and lungs. In a concentrated form it is probable that from two to six ounces of alcohol would prove fatal.

Period of death.—In poisoning by alcohol, death may take place in a few minutes, or not until after the lapse of several days. The shortest fatal case which I have found reported, excepting the instance quoted from Orfila (p. 635), was that of a man who died in half an hour after swallowing a bottle of gin for a wager. This occurred in London, in 1839: in a quarter of an hour after taking the gin he appeared intoxicated; he soon became insensible, and died in ***half an hour***, although a large quantity of the spirit had been in the meantime removed by the stomach-pump. In general, if the case proves fatal, death takes place within twenty-four hours. Alcohol, it must be remembered, may destroy life indirectly, *i. e.* by exciting an attack of congestive apoplexy in those who are predisposed to this disease, and thus a small quantity may accelerate death.

Treatment.—The contents of the stomach should be withdrawn by the pump as speedily as possible. Cold affusion, if the surface be warm, or, as suggested by Dr. Christison, the injection of cold water into the ears, may serve to rouse a person. Death may take place even when the stomach has been emptied, but this affords commonly the only chance of saving life. The vapor of ammonia may be employed as a stimulant, and bleeding may be resorted to if there should be great cerebral congestion. Bleeding should in any case be employed with great caution, as it is apt to depress the vital powers and diminish the chance of recovery. The electro-magnetic apparatus may be used as in poisoning by opium; but it is necessary to remember that keeping a person roused, does not aid recovery so long as the poison is allowed to remain in the body.

Analysis.—The contents of the stomach in a rapidly fatal case will have the odor of alcohol, or of the alcoholic liquid taken. The odor is not always perceptible, or it may be concealed by other odors. In a case of poisoning by gin, the liquid drawn from the stomach by the pump in seven hours had no odor (p. 636).

In order to detect alcohol, the *whole* of the contents or of the suspected liquid should be placed in a capacious retort and distilled in a water-bath, with a proper condensing apparatus attached. If the suspected liquid should have an acid reaction, it should be first neutralized either by a solution of potash, or of carbonate of potash, or soda. The watery liquid obtained should be mixed with chloride of calcium or anhydrous sulphate of copper, in sufficient quantity, and submitted to a second distillation, in a smaller retort, in a water-bath. The liquid obtained by the second distillation should be agitated with rather more carbonate of potash than it will dissolve, in a small tube provided with a stopper, and allowed to stand. A stratum of alcohol, if present, will, after a time, float on the surface, and may be drawn off by a pipette and examined. *Tests.*—1. Alcohol has a hot pungent taste, a peculiar odor, and is very volatile. 2. Absorbed in asbestos, it burns with a pale blue flame, which deposits no carbon on white porcelain; and when burnt in the mouth of an inverted test tube, containing a few drops of solution of baryta, it produces a well-marked deposit of white carbonate of baryta. Lime water may be substituted for baryta in this experiment. Carbonic acid and water are the sole products of its combustion. 3. It dissolves camphor. 4. It sets free green oxide of chromium when boiled with a few drops of a saturated solution of bichromate of potash mixed with sulphuric acid. (Dr. Thomson, in Monthly Jour. Med. Science, Dec. 1846, p. 412.)

Objections.—Ether, pyroxylic spirit, and methylated spirit produce the same results with some of the tests (2, 3, 4). Ether is known by its odor and the yellowness of its flame when burnt on asbestos, as well as by a smoky deposit on white surfaces. Pyroxylic spirit is also known by its powerful and peculiar odor. It reduces chromic acid to oxide of chromium (4) in the cold. It burns with a smoky flame. Methylated spirit is alcohol containing about ten per cent. of pyroxylic spirit.

forwards to the junction of the neck with the body of the retort. As alcohol is volatilized before the water, so soon as drops of liquid begin to condense about the neck of the retort, it will be observed, if alcohol is present in the vapor, that the litmus paper at the further end of the tray will be reddened (owing to the production of acetic acid), while the paper nearer to the body of the retort will remain blue. If any acid should be distilled over in the vapor, this paper will also be reddened: but as the liquid or solid, moistened with water, should be previously neutralized by potash, the escape of acid vapors will be thereby prevented. If no reddening of the paper occurs, no alcohol is present: if it is rapidly reddened, the tray may be removed and the liquid product collected by the usual process of condensation in a cool receiver. It may be then rectified by carbonate of potash, and examined by the tests described p. 641. This is, therefore, an addition to the usual process. Excepting alcohol, ether, or wood-spirit, no liquid is known which will produce this effect with platina-black. The two last may be identified by the odor. The ordinary products of putrefaction do not prevent this conversion of alcohol to acetic acid.

This process is open to the objection that ether and pyroxylic spirit will produce similar effects: hence it does not present any advantage over the chromic process above described (p. 641), except under one condition. If the contents are putrefied, sulphuretted hydrogen may be evolved, and reduce the chromic acid: but it will produce no fallacy with the platina-black. To guard against this source of error in the chromic process, it will be necessary to test the vapor by paper impregnated with a salt of lead.

Poisoning of alcoholic liquids.—Instances have occurred in which alcoholic liquids have been made the vehicles for administering powerful poisons, such as opium, prussic acid, stramonium, tobacco, nux vomica, or cocculus indicus. Persons have been thus rendered insensible; and in this state have been robbed or murdered. Such cases may commonly be recognized by the fact that the symptoms, when known, are of far too severe a character to be referable to the small quantity of alcoholic liquid taken. Tincture of opium is not unfrequently administered in this way: and in such a case there may be some difficulty in deciding whether the symptoms of intoxication were due to the drug or to the spirit. In such cases alcohol may be found in the stomach, although not the cause of death.

ETHER.

Ether vapor. Symptoms.—It has been long known that the vapor of ether acts on the brain and nervous system like a powerful narcotic. Orfila mentions the case of a young man who was thrown into a state of insensibility by reason of his having respired ether vapor. He remained for several hours in an apoplectic condition, and would have died but for his removal to fresh air and the application of proper means for his recovery. (Toxicologie, éd. 4ème, 1843, ii. 532.) Dr. Christison quotes a similar instance in which a female was found lying in her bed quite dead, in consequence of her having respired, during the night, an atmosphere strongly charged with ether vapor. On inspection, the stomach was found reddened internally, and the lungs were gorged. (Op. cit. 965; also, Ed. Med. and Surg. Journal, xxxv, 452.) The poisonous effects of the vapor have been therefore known for a long time, although the attention of the profession has been only of late years particularly drawn to the subject.

The vapor of ether is very heavy (sp. gr. 2·58), and possesses a strong odor at all temperatures. It is diffusible and volatile, properties which are more favorable for the operation of this liquid in the state of vapor, than for the action of alcohol.

When the vapor is respired, it enters the blood in the pulmonary vessels, and the effects are almost immediate. The person falls into a lethargic condition, the breathing becomes slow, deep, and stertorous, the skin pale and cold, the face livid, the lips assume a darker hue, the pulse is quickened, the eye is glassy and the pupil dilated and fixed: but the late Dr. Snow found the eye sensible to light in all the stages of etherization: the muscles of the body are flabby and relaxed. A small quantity of ether introduced into the blood through the lungs, produces these striking symptoms in from two to four minutes; and if fresh air be substituted as soon as unconsciousness begins, they disappear just as rapidly. In a more advanced stage the pulse slackens, and the temperature of the body rapidly falls. Half an ounce of ether, or even less, inhaled in the form of vapor, would produce a much more powerful effect on the system, than one or two ounces taken into the stomach as a liquid. The sudden cessation of the symptoms, and the restoration of sensibility, are owing to the rapid elimination of the vapor through the lungs. If the respiration of the vapor be prolonged for from ten minutes to half an hour, there is coma, the pulse sinks, and there is some difficulty in rousing the person. The after effects are also more serious,—there is exhaustion, a feeling of stupefaction, with other unpleasant narcotic symptoms: but occasionally the patient has fallen into a quiet sleep. The most remarkable effect in those who suffer under this form of poisoning is the apparently complete paralysis of the nerves of sensation: for the most painful operations have been often borne by persons in this state without any consciousness of pain. In some instances, unpleasant sensations are stated to have been experienced, probably the result of association, for no consciousness whatever has existed that an operation had been performed. Another singular fact is, that although there is a general relaxation of the limbs, there is still a power of moving them, and the senses have been preserved while general sensation has been lost. It is also remarkable that the involuntary muscles do not partake of this relaxation. The vapor has varied in its effects according to habit, idiosyncrasy, and other conditions. Some persons appear to have suffered no particular symptoms: it has failed to throw them into a lethargy. This has probably arisen from its having been imperfectly breathed: others have suffered from irritation of the lungs, some have become intoxicated, and others, again, have become so excited by it, as to require forcible control. In young persons, nausea and vomiting have been noticed among the symptoms. As a general rule no dangerous effects appear to have followed the respiration of this vapor for surgical purposes; but this inference has been chiefly drawn from those cases in which it had been cautiously administered for a short period; and probably there was no tendency to congestion of the brain or lungs. The narcotic effects are produced in three, and on an average in five minutes. In cases of prolonged respiration of the vapor, serious symptoms, and even death, have resulted. Dr. Bigelow found that a young man who respired ether vapor for thirty-five minutes, nearly sank under the effects. The pulse fell, the respiration became slow, the skin cold, and there was the most perfect insensibility. Cold affusion was employed for ten minutes without effect, and the ambulatory treatment, adopted in poisoning by opium, was then resorted to. In about half an hour he was able to lift up his head; but he did not recover his consciousness for an hour. (Lancet, Jan. 2, 1847, p. 7.) As in the case of all aerial poisons, the protracted respiration of ether vapor must tend to render recovery difficult, by thoroughly impregnating the blood with the poison. Large dogs have been observed to lose the power of sensation in eight minutes: and they died if the action of the ether was continued for forty-five minutes.

Appearances.—In the case of a man who died in about ten minutes from the effects of the vapor, on inspection twenty-two hours after death, the brain, lungs, heart, kidneys, and spleen, when cut into, gave out a strong odor of ether. The blood in the vessels was of a very dark color, liquid, and of a viscid character. The posterior part of the lungs was strongly congested with dark-colored blood; and in the anterior portion of the organs a frothy mucus was found filling the air-tubes. The mucous membrane of the whole of the air-passages was deeply injected (Med. Gaz. vol. 41, p. 432.) On examining the bodies of animals thus poisoned, the principal appearances have been great congestion of the vessels of the membranes (pia mater) and of the sinuses of the brain, the substance being but little altered. The vessels of the upper part of the spinal marrow have been observed to be especially distended with dark-colored blood. Both sides of the heart have been found filled with dark blood; the liver and kidneys gorged; the spleen not always congested: the blood black and liquid throughout the body. The cause of death in these cases may be assigned partly to the want of aeration of the blood by oxygen, and its accumulation in this impure state in the brain; and partly to a directly poisonous action of the absorbed vapor, only manifested by its employment for a long period. In order to prevent this, it has been advised to allow the patient to breathe air occasionally, and to alternate the respiration of pure air with that of the vapor; but, unless there is a complete restoration of sensibility and consciousness, the poison must go on accumulating in the system, and if the person be allowed to recover thus completely, it may be regarded as a commencement of its poisonous action *de novo:* if not thus allowed to recover, he is in danger of sinking under its effects. The continued administration of morphia or strychnia, at intervals so short as not to allow of a recovery from each successive dose, must cause an accumulation in the system, and lead to fatal results. It is so with ether vapor, and experience now points to the propriety of withdrawing its use altogether, in those cases in which the administration of it would require to be protracted for a long period in order to produce narcotic effects. The fact that hundreds have recovered without ill effects during its temporary employment for the extraction of teeth, or similar operations, has of course no bearing on this question. A man may breathe a mixture of carbonic acid or sulphuretted hydrogen with air for a few minutes: but he would die if he was compelled to respire it for half an hour longer. The vapor is so insidious in its operation that it may be respired during natural sleep without rousing the individual, and there is no doubt that it might thus be used as a ready means of destruction for the young and the aged. (Gaz. Méd. Sept. 11, 1847, p. 725.)

In a case reported in the Medical Gazette, the death of a man was occasioned by the respiration of the vapor at intervals, for a period of only ten minutes during an operation. He recovered from the comatose effects; but there was no tendency to reaction, and he gradually sank, and died on the second day. It was remarked in this case that there was great flaccidity and general relaxation of the muscular system, and the arteries which were divided during the operation (lithotomy) appeared to have lost all their contractile power. On inspection, there was membranous congestion of the brain; the lungs were engorged at the back part; the heart was flaccid, of a natural size, and nearly empty; the left kidney pale, the right congested. The blood was perfectly fluid. (Med. Gaz. vol. xxxix, 414.) A similar case was privately communicated to me by an eminent London surgeon, in which he stated that there could be no doubt of the vapor having been the direct cause of death. The patient sank after the operation, under symptoms which in similar circumstances he had never before witnessed. In the Medical Gazette there is reported another case (vol. xxxix, p. 585) in which the vapor was administered to a female for a period of thirty-five minutes. She recovered her senses, but did not rally from the operation. She complained of numbness in the feet and legs, and the secretions were suspended. She died the following day. On this occasion the vapor appeared to induce a perfect state of paralysis of the brain and nervous system. On inspection, the lungs were slightly congested posteriorly; the heart was flabby, and contained less blood than usual; the brain was healthy, its membranes were rather congested, and the blood was generally in a liquid state. In a case that occurred to Mr. Eastment, there was no disease, nor any particular state of the body to account for death. Amputation was performed for compound fracture of the thigh; the ether was inhaled by the patient, a boy æt. 11, for about ten minutes. After the operation he was not only greatly exhausted, but in a state of apparent intoxication. There were alternate manifestations of excitement and depression of the sensorial powers, at one time resembling delirium, at another syncope, and again passing into violent intoxication, until death took place three hours after the operation. (Med. Gaz. xxxix, 632.) The symptoms were here such as might be expected from the poisonous effects of ether, and unlike those which usually attend collapse from an operation.

These facts, then, show that the respiration of this vapor, even for so short a period as ten minutes, may be in some instances attended with fatal consequences. Whether the vapor was properly administered or not, is, in relation to legal medicine, not so much the question as whether it caused death! In any case, the inhalation of this vapor must be looked upon as temporary poisoning, with, *cæteris paribus*, a better chance of recovery than exists in most other instances of aerial poisoning.

Ether is said to produce its narcotic effects, when administered as a vapor by the rectum, without the production of those distressing symptoms which often accompany the first attempts at breathing it. (See Med. Gaz. xxxix, 950.)

Liquid Ether. Symptoms and Effects.—The effects produced on the system by *liquid* ether, are not unlike those occasioned by alcohol. Orfila found that about half an ounce of sulphuric ether, administered to a dog, caused, in a few minutes, a disposition to vomit. This was followed by giddiness, and in ten minutes by an entire loss of power in the muscles. The breathing was painful and hurried, but there were no convulsions. After a slight abatement in the symptoms, the dog fell into a state of insensibility and died in three hours. The whole of the mucous membrane of the stomach was of a blackish-red color, and with the other coats intensely inflamed. There was slight inflammation of the duodenum; but the rest of the alimentary canal was in a healthy condition. The heart contained black blood partly coagulated: the lungs were gorged with fluid blood. (Op. cit. ii, 531.)

Ether in moderate doses, has a hot burning taste, and produces during swallowing a sense of heat and constriction of the throat. It causes, like alcohol, great excitement and exhilaration, with, subsequently, intoxication, but persons may become habituated to it; and thus, after a time, it may be taken in very large quantities with comparative impunity (see p. 89). The medicinal dose is from half a drachm to two drachms. Dr. Buchanan has known seven drachms of it taken at once: it produced at the pit of the stomach an uneasy sensation of heat and pain, which only the callous stomach of a dram-drinker could withstand. (Med. Gaz. xxxix, 715.) In 1845, a young man was brought before one of the London police-magistrates, in a stupefied state: to those present he appeared to be intoxicated. It was proved in evidence that he was in the habit of taking ether, and that he was then laboring under its effects. It appears that he frequented the shops of druggists, and swallowed this liquid in large doses.

Fatal dose.—There is no instance reported of ether having caused death when taken in the liquid form; but it has never been swallowed at once in a large dose like alcohol. It does not admit of dilution with water to the same degree as alcohol, and therefore it acts, *cæteris paribus*, as a more violent local irritant. It requires ten parts of water to dissolve one of ether; hence, unless, as Dr. Buchanan has remarked, the water is in very large proportion, it does not render the ether palatable to most persons. It is at present impossible to give any precise opinion respecting the smallest quantity of this liquid which would destroy the life of an adult. The quantity of ether *vapor* required to produce complete insensibility varies from six drachms to one ounce in the adult, and in children in the same proportion according to their size. The quantity required to keep up insensibility is seldom greater than that which induced the state of etherization.

Treatment.—In reference to the vapor—the failure of the pulse, with stertorous breathing, frothing at the mouth, and great lividity of the face, are signs of danger. The ether should be withdrawn, the face and neck exposed to a free current of air, and cold water dashed upon the skin, although, from the suspension of sensibility, stimulants produce little effect. Artificial respiration should be resorted to when the breathing has ceased.

Analysis.—Ether is at once identified by its powerful odor, even in the smallest proportion. 1. It is highly inflammable, and burns with a yellow smoky flame, producing carbonic acid and water. 2. When shaken with its bulk of water, only a small portion is dissolved, the rest floats on the surface. If taken in the liquid form, it may be separated from the contents of the stomach by distillation, and the product rectified by redistillation with carbonate of potash at a temperature of about 120°.

Hoffman's liquor is a mixture of alcohol and ether. This may be easily examined by agitating it with half its bulk of water; the ether (beyond about one-tenth of the quantity of water used) rises to the surface and may be drawn off by a pipette. The alcohol is dissolved and retained by the water; this liquid may be procured by distillation with carbonate of potash or fused chloride of calcium, and its properties then tested.

Organic liquids. The tissues.—When death has taken place from ether vapor there is a strong odor throughout the body, if the examination is recent. The quantity absorbed by the blood is small, and it is probably partially converted to aldehyde in that liquid. There can be but little hope of success in attempting to procure it from the blood or the soft organs by distillation, although M. Flandin states that he has extracted it from the blood. Whether ether be taken in the form of liquid or breathed as vapor, there is no doubt that it is absorbed and circulated with the blood; in the latter state, with great rapidity. M. Amussat noticed in his experiments on animals, that after prolonged inhalation the arterial blood lost its red color, and became black. The bright arterial tint was, however, soon resumed on suspending the process. Ether, besides rendering the blood black, causes it to become more liquid. The change in this fluid is very much like that which is observed in fatal cases of asphyxia. (Gaz. Med. Sept. 11, 1847, p. 725.)

When ether has been taken as a liquid and has caused death, it may be separated from the contents of the stomach by the process described for alcohol (ante p. 641). The chromic process applied to the vapor during distillation will enable the analysis to detect a minute quantity, and the odor may be easily distinguished from that of alcohol or pyroxylic spirit.

CHLOROFORM.

Chloroform vapor. Symptoms.—The symptoms which the vapor of chloroform produces are very similar to those produced by the vapor of ether; but the person passes much more rapidly into a complete state of insensibility and general relaxation. From being at first excited, he becomes motionless,—the pupils of the eyes are widely dilated,—the sphincters lose their contractile power,—the face is pale, sometimes livid; the lips are congested, the breathing is slow, after a time stertorous, the surface cold, and the pulse gradually sinks. There is an entire loss of sensibility. The sinking of the pulse in some cases is so sudden as to expose the patient to death by syncope (Med. Gaz. xl. 1036). In other instances violent convulsions have supervened, even when the dose has been only from half a drachm to a drachm. These effects may be occasionally aggravated by idiosyncrasy, or by latent organic disease. The fatal effects of the vapor are likely to be manifested when it is breathed rapidly and unmixed with air. There is no doubt that in a concentrated state chloroform is a powerful cerebral poison. It is absorbed into the blood, which it darkens, as in cases of asphyxia, and is thus circulated throughout the system. The blood is probably directly poisoned by it. The effects produced by this vapor come on insidiously; a person who breathes it in the absence of assistance is thus rendered powerless, and may die with the mouth over the inhaler. Several deaths of this accidental kind are recorded. (See Med. Times and Gazette, Nov. 21, p. 533.)

Appearances.—Congestion of the vessels of the brain and its membranes has been met with, but not uniformly; the lungs congested, or in an apoplectic condition; the heart flaccid, and the cavities frequently empty, or containing but little blood; in some cases the right cavities have been found greatly distended; the blood generally dark in color, and very fluid, sometimes mixed with air-bubbles. These are the principal appearances. Among fifty fatal cases recorded by Dr. Snow, it appears that thirty-four inspections were made. In three the state of the lungs is not mentioned; in four they are said to be normal, and in twenty-seven there was engorgement of the lungs, or of the right side of the heart: in the majority both these conditions were met with (Med. Times and Gazette, Oct. 23, 1858, p. 431). In some cases the cavities of the heart have been found empty. Congestion of the liver, spleen, and the kidneys is not unusual.

Fatal cases have been proportionally much more numerous from the use of chloroform vapor than from ether vapor. In some of these, latent morbid conditions of the heart or brain may have led to the unfortunate result; in others, the improper mode of administering the vapor. But there have been undoubtedly cases in which, with the exercise of proper skill and care, death has still occurred. A girl, æt. 15, had the vapor administered to her on a warm cloth. The quantity of chloroform used was about one drachm. In about half a minute, she became insensible; the lips were suddenly blanched, and it was observed that one arm was rigid. The respiration was quick, but there was no stertorous breathing. The patient suddenly spluttered at the mouth, as if in an epileptic fit. Attempts were made to revive her, but she was dead in from two to three minutes after commencing the inhalation. On inspection, the lungs were found greatly congested. There was a bloody froth in the air-passages, mixed with mucus: the epiglottis was reddened. The brain and its membranes were more congested than usual, and the ventricles contained more than the usual quantity of serum. The abdominal viscera were also highly congested. (Med. Gaz. xli, 250–254.) These appearances correspond to those observed in the action of the vapor on animals. In a case that occurred to Dr. Jamieson, in which the quantity inhaled was probably three or four drachms, the appearances of general congestion were similar (Med. Gaz. xli, p. 318); it was remarked that the mass of blood was darker than natural, and that it was fluid and unusually thin. The muscles were also darker than ordinary.

As in cases of alcoholic poisoning, death may take place from secondary causes after apparent recovery. A gentleman, æt. 49, had the vapor administered to him to the amount of six drachms, for the purpose of a surgical operation. The state of insensibility was continued during eight minutes. Consciousness was completely restored after the operation, and the patient conversed freely. In the course of the day he experienced unpleasant sensations in his head, and he passed a restless night. The next morning the pulse was 100, and rather full, and he seemed drowsy. In the afternoon he became comatose, and in spite of treatment he died about forty hours after the administration of the chloroform. On inspection twelve hours after death, there was great congestion of the brain, with some effusion of serum. It is probable that in this case death was accelerated, if not caused by chloroform. (Kesteven's Quarterly Report on Toxicology, Med.-Chir. Rev. April, 1854, p. 582. Case by Dr. King, in Ed. Med. and Surg. Journal, Jan. 1854.) Another case of death subsequent to apparent recovery has been reported by Mr. Lane, Surgeon to the Lock Hospital. (Med. Times and Gaz. June 3, 1854, p. 572.) The patient was a youth, æt. 18. Chloroform was administered for the purpose of preventing pain during an operation. About *two drachms* were inhaled through a simple mouth-piece, by which the nostrils were left uncovered. After about six minutes, insensibility appeared to be coming on; the pulse was then of a good volume. There was nothing to indicate impending danger, when, after a few more inhalations, the pulse suddenly failed, became quite imperceptible, and the countenance assumed a pale and leaden hue. The inhalations were stopped, and attempts at resuscitation were made. The pulse returned, as well as spontaneous breathing, and the countenance assumed a slight flush. In ten minutes these favorable symptoms ceased, the countenance became deathlike, and the patient sank rapidly. On inspection the next day, there was great venous congestion in the brain and lungs. The heart was slightly enlarged, and the walls of the ventricles were thinned. The blood was of a dark color, and universally fluid. There appears to have been nothing in the condition of this patient to account for the fatal effects of the vapor. (See also L'Union Medicale, Sept. 3, 1857, No. 106.)

Numerous fatal cases from the effects of chloroform vapor have been reported in various medical periodicals. The symptoms and appearances have varied but little from those above described. As it would be impossible within the limits of this work to give any detailed account of the cases, I must refer the reader for additional information on the effects produced by this powerful agent, to the accurate reports of a few selected cases. (See Med. Times and Gazette, Oct. 9, 1852, p. 361; April 9, 1853, p. 369; August 13, 1853, p. 173; Oct. 15, 1853, p. 407; Oct. 22, 1853, p. 422; Oct. 29, 1853, p. 461; Nov. 26, 1853, p. 562; Lancet, June 4, 1853, p. 523; Oct. 29, 1853, p. 410; Edin. Monthly Jour. April, 1850, p. 377; 1855–6, i, p. 524. See also communications by Dr. Snow, to the London Journal of Medicine, April, May, and June, 1852; and Association Med. Jour. Feb. 11, 1853, p. 131.) These cases are well calculated to show that even in the hands of experienced persons, and when all reasonable precautions are taken, chloroform vapor may in a sudden and unexpected manner exert a fatal influence by suspending (paralyzing?) the action of the heart, or disturbing the functions of the brain and lungs. According to Dr. Snow, the recorded cases of death from the inhalation of the vapor, up to 1853, amounted to thirty-seven. (Med. Times and Gazette, Dec. 24, 1853, p. 665.) But he has since recorded the deaths of thirteen. Fatal cases still occasionally present themselves in spite of every precaution, and there are probably some the details of which do not come before the public or profession. The result of experience is, that in certain morbid conditions of the heart and brain the inhalation of this vapor even in ordinary doses is likely to prove fatal; and we further learn, from the examination of the fatal cases, that the morbid states of these organs which thus render the use of chloroform vapor dangerous, are not always ascertainable during life. In consequence of the effects of this vapor in rendering persons insensible and incapable of offering resistance in assaults with intent to commit rape and other felonies, the using of it for such a purpose has been made a felony by Act of Parliament (14 and 15 Vict. ch. 19, s. 3).

A mixture of four parts of ether to one part of chloroform has been much used in the United States as a substitute for these liquids. One drachm of this mixture administered with due precaution (in vapor), caused death in a few minutes. (Med. Times and Gaz. Aug. 22, 1857, p. 198.)

Liquid chloroform.—This liquid, when taken in a large dose, appears to affect the system like alcohol, but as a *liquid* it cannot be regarded as an active poison. I have elsewhere recorded a case communicated to me by Mr. Jackson, of Sheffield, in which a man swallowed *four ounces* of chloroform. He was able to walk for a considerable distance after taking this dose, but he subsequently fell into a state of coma—the pupils were dilated, the breathing stertorous, the skin cold, the pulse imperceptible, and thére were general convulsions. He recovered in five days. (Med. Gaz. vol. xlvii, p. 675.) A private in a cavalry regiment in the United States swallowed nearly two ounces of chloroform. He was seen ten or fifteen minutes afterwards; he had already vomited, and was found insensible, with stertorous breathing, and a pulse of about 60. The stomach-pump was employed, and some spirits of ammonia injected. The pulse became more feeble, the breathing slower, and the pupils were insensible to light. The surface became cold, and for a time he continued to get worse, the face becoming purple, while the pulse was intermittent, and hardly discernible. Two hours and a half after taking the poison, however, a gradual improvement commenced, but sensibility did not return until four hours later. For several days he continued to suffer from great irritability of the stomach, and eventually he had an attack of jaundice. (Amer. Jour. Med. Science, Oct. p. 367; Med. Times and Gazette, Nov. 28, 1857, p. 558.) In two cases alarming symptoms have been produced by much smaller doses, and one of these proved fatal. In

March, 1857, a lady swallowed half an ounce of pure chloroform. In five minutes she was quite insensible, generally convulsed, the jaws clenched, the face slightly flushed, the pulse full and rather oppressed, and she foamed at the mouth. She vomited, and in twenty minutes the convulsions had left her; soon afterwards she had a relapse, and did not recover for twenty-four hours. (Med. Times and Gaz. Dec. 12, 1857, p. 615.) The symptoms in this case appear to have been mixed with those of hysteria and epilepsy. The following case communicated to me by Mr. Thursefield, of Brosely, in March, 1854, appears to be a clear case of poisoning by liquid chloroform. A boy, æt. 4, was brought to him by his father in a state of total insensibility. It appears that he had swallowed a *drachm* of chloroform, and soon afterwards laid his head on his mother's lap and lost all consciousness. Mr. Thursefield saw him about twenty minutes afterwards. He was then insensible, cold, and pulseless. Mustard plasters were applied to the legs; they acted well, but produced no impression on the sensibility. His breathing varied; it was sometimes natural, at other times stertorous. He became warmer, his pulse full and regular; and he continued *three hours* in this state, when he died quite calmly, without a struggle, in spite of every effort made for his recovery.

Fatal dose. Period of death.—In some cases death has taken place within two minutes from the commencement of inhalation. In one instance, in which only thirty drops had been inhaled in vapor, the patient died in one minute, and in another so small a quantity as fifteen or twenty drops proved speedily fatal. (Table of fatal cases by Dr. Warren, U. S. p. 23.) Chloroform, therefore, operating through the lungs, has destroyed life more rapidly, and in a smaller dose, than any other poison known. Its fatal effects are sometimes suddenly produced after the withdrawal of the vapor. In one case, witnessed by a friend, the heart suddenly ceased to beat four minutes after the vapor had been withdrawn. The digital arteries which had been divided in the operation, suddenly ceased to bleed. The man was dead. The smallest dose which has proved fatal in the liquid state, was in the case of the boy (supra), who died in three hours. A medicinal dose of liquid chloroform varies from five to twenty minims or more, and the quantity in the form of vapor varies in an adult from one to four drachms, and even more, according to the effects produced, the state of health, and other circumstances. In some cases a large quantity is borne with impunity. Mr. Garner states that a lady breathed in his presence the vapor of more than an ounce and a half, and he ascertained that she had used for the relief of pain, as much as sixty-two ounces of liquid chloroform in vapor in twelve days. (Med. Times and Gazette, Nov. 22, 1856, p. 528.) In certain forms of disease (tetanus), large doses may be borne. In a case at Guy's Hospital, more than a pint of chloroform was used in twenty-four hours. There is no doubt that the vapor is rapidly eliminated by the lungs, and unless the blood be overcharged at any one time, there appears to be no danger from these large doses. An instance of the remarkable uncertainty in the effects of chloroform vapor is furnished by a case which occurred to Dr. Roberts of Edinburgh. A lady, æt. 36, who had frequently before taken chloroform without injury, insisted upon having it administered to her previous to the extraction of a tooth. A quantity of pure chloroform, equal to about one drachm and a half, was poured from a bottle; she made about nine or ten inspirations of the vapor, and when in the act of asking for more chloroform, she suddenly gave a convulsive start, made a stertorous inspiration, and with eyes and mouth open, sank to the floor. Every effort was made to restore her, but without effect. The principal appearances met with after death, were congestion of the capillaries of the abdomen, the heart was smaller and its muscular substance thin, the right cavities were gorged with dark blood, the lungs were congested posteriorly, and the vessels of the brain were also congested. (Ed. Med. Jour. Dec, 1855, p. 524.)

Treatment.—As death frequently takes place suddenly from a suspension of the action of the heart (syncope), the treatment to be pursued after the withdrawal of the chloroform vapor, consists in a free exposure of the face to a current of air, cold affusion, compression of the chest, and the maintenance of respiration by artificial means. The employment of stimuli, externally or internally, can be of little benefit, as sensibility is paralyzed by the poison. There can be no antidote to a poison which is diffused through the whole of the blood. The only source of elimination is through the lungs; hence, in poisoning by ether vapor, the only chance of restoration is in maintaining the act of breathing. The respiration of oxygen is not likely to be of practical benefit. The patient will either have recovered or died before this can be used. In cases of an asphyxial kind, in which the heart continues to beat after respiration has ceased, there is great hope of recovery by resorting to artificial respiration, by the prone and rotation treatment recommended by the late Dr. Hall. (Med. Times and Gaz. April 7, 1858, p. 416; also June 26, p. 662.) In poisoning by liquid chloroform, the stomach-pump should be employed.

Analysis.—Chloroform is a heavy, colorless liquid (sp. gr. 1·484), neutral in its reaction, sinking in water in rounded globules, and only to a slight extent dissolving in that liquid. It has a fragrant odor like that of apples; it is dissolved by alcohol and ether. It is very volatile, but not combustible. Like alcohol, it dissolves camphor. Nitric and sulphuric acids produce no change in it. It boils at 140°, and evolves a vapor which at a red heat is resolved into chlorine and hydrochloric acid. On this effect a process has been suggested for separating it from the *blood and tissues* when it has proved fatal in the form of vapor. The liquid or solid supposed to contain chloroform, is placed in a Florence flask, to the neck of which is fitted a cork, perforated to admit a German glass tube of about one-quarter of an inch in diameter, and from one to two feet long. The flask is heated by a water-bath. By wrapping wet filtering paper round the portion of this tube near the flask, the vapor may be condensed, and the chloroform collected as a liquid. To test the decomposed vapor, a piece of paper wetted with a solution of starch and iodide of potassium is introduced into a cool part of the tube, or exposed at the open end, and a portion of the tube between this point and the flask is then strongly heated by a spirit lamp. If chloroform be present chlorine is evolved, and the paper is rendered blue. If the chlorine comes over in large quantity, the blue color is produced and rapidly destroyed. If the starch paper should be placed in a warm part of the tube or receive the heated vapor, the color will not appear, so that the operation requires care. The decomposed vapor received on litmus paper will first redden and then discharge the color. It will also produce a white precipitate, with a solution of nitrate of silver (from chlorine, and hydrochloric acid evolved). In order to guard against any fallacy from the escape of acid vapors by distillation, the liquid should be previously neutralized by potash. The late Dr. Snow and myself examined by this process the blood of a boy, who had died in Guy's Hospital from the effects of chloroform vapor, but without detecting any trace of this substance. There was no odor in the blood, and the result was negative. In Dr. Jamieson's case (ante, p. 649), the blood did not smell of chloroform, but the bloody fluids yielded, on distillation, a small quantity of a volatile liquid, which had a strong chloroform smell.

As chloroform is much more volatile than ether, and its odor is not so pungent, it is not so easily detected in the dead body by the smell. The body should be inspected as soon as possible, and any solids or liquids intended for examination should be placed in well-closed glass vessels. When chloroform has been taken as a *liquid*, it may be separated from the contents of the stomach by a water-bath distillation at 120°. Any water that may come over with it, may be separated by redistillation with chloride of calcium. It may be known by the properties described above, also by administering the vapor to a small animal.

AMYLENE.

The vapor of this anæsthetic agent was introduced by the late Dr. Snow as a substitute for the vapor of chloroform. It has been found to produce a loss of sensibility without causing complete coma or stupor. Its use has already led to at least two deaths; and on the whole, it does not appear to be so safe an agent as chloroform vapor for surgical purposes. The only *appearance* met with was in one case an emphysematous state of the lungs or excessive dilatation of the air-cells (Med. Times and Gazette, April 4 and 18, 1857, pp. 332, 381); and in the other, a distension of the right cavities of the heart with dark fluid blood. There was no congestion of the brain, and no smell of amylene perceptible in the body. (Med. Times and Gazette, Aug. 8, 1857, p. 138.)

CAMPHOR.

There is only one instance recorded in which camphor has proved fatal in the human subject: but it has on several occasions produced alarming symptoms, and would probably have destroyed life, had it not been early removed from the stomach. In the few cases that have been observed, its effects were somewhat different, although both in man and animals they were referable to an impression on the brain and nervous system.

Symptoms and appearances.—The following case is reported by Mr. Hallet, of Axminster. A woman swallowed in the morning about *twenty grains* of camphor dissolved in rectified spirits of wine and mixed with tincture of myrrh. In half an hour she was suddenly seized with languor, giddiness, occasional loss of sight, delirium, numbness, tingling, and coldness of the extremities, so that she could hardly walk. The pulse was quick and respiration difficult, but she suffered no pain in any part. On the administration of an emetic, she vomited a yellowish liquid, smelling strongly of camphor. In the evening, the symptoms were much diminished, but she had slight convulsive fits during the night. The next day she was convalescent; the difficulty of breathing, however, continued more or less, for several weeks. This is the smallest dose of camphor which appears to have been attended with serious symptoms. A man, æt. 39, swallowed about *thirty-five grains* of powdered camphor, prepared for lozenges. In twenty minutes, giddiness and dimness of sight came on; and he fell from a chair in a kind of epileptic fit, which lasted about ten minutes. The limbs were cold, the pulse was frequent and scarcely perceptible: when roused, he had scarcely power to articulate. A quantity of a clear liquid, smelling strongly of camphor, was drawn off by the stomach-pump. The man did not recover for a week, suffering chiefly from general exhaustion and suppression of urine: this latter symptom continued more or less for three months afterwards. There was no disorder of the stomach or bowels. Dr. Christison refers to a case in which thirty grains of camphor given in an injection produced numbness of the scalp and other nervous symptoms. In two other instances mentioned by him, in each of which forty grains have been taken, the symptoms were giddiness, chilliness, convulsive fits, and delirium. In larger doses, symptoms of irritation make their appearance. Dr. Siemerling, of Stralsund, relates, that a man, æt. 69, swallowed 120 grains of camphor, in order to relieve some rheumatic symptoms under which he was laboring. When seen three hours afterwards, he resembled a drunken person. He complained of burning heat in the mouth, throat, and stomach,—throbbing in the head, pains in the course of the spine, and a ringing in the ears,—with the appearance of a dazzling light before the eyes: these symptoms were followed by convulsive twitchings and insensibility. In this state he continued for an hour and a half, perspiring profusely. The man slowly recovered; but none of the camphor appears to have been ejected from the stomach. (Wildberg's Jahrbuch, 1837, 3 B. 4 H.) Three cases of poisoning by camphor are reported by Dr. Schaaf, one of which proved fatal. A woman gave about thirty grains (half a teaspoonful) of powdered camphor to each of her three children as a vermifuge. Two of the children were respectively of the ages of three and five years, the third was an infant aged eighteen months. The first symptoms were paleness of the face, with a fixed and stupid look. Delirium followed, with a sense of burning in the throat, and great thirst. Vomiting, purging, and convulsions supervened, and in one child the convulsions were most violent. The two elder children, after suffering thus for three hours, fell into a comatose sleep, and on awaking the symptoms passed off. The infant died in seven hours, not having manifested any return of consciousness from the first occurrence of convulsions. (Med. Gaz. xlvii, p. 219.) In this Journal, the dose is erroneously stated to be 180 grains. For a report of the case, see Journal de Chimie Médicale, 1850, p. 507. The severity of the symptoms is fully explained by the large quantity administered and the age of the children. In a dose of one drachm given in a clyster, camphor produced alarming symptoms. (Med. Gaz. xlviii, 552.) In a case, reported in the Medical Gazette (vol. xi, 772), 120 grains were taken by a physician, and all that he experienced was, lightness in the head with great exhilaration. There was no derangement of the stomach or bowels. He slept profoundly for some hours, and awoke very weak and exhausted. He also perspired greatly during his sleep. It is difficult to draw any conclusion from this case, as the quantity taken was conjectural; and the patient was not seen by any person while laboring under the effects of the poison.

A case of poisoning by camphor would at once be recognized by the odor of the breath,—a symptom, which would attract the attention of a non-professional person. A woman took, by mistake, half an ounce of camphor dissolved in olive oil. She did not discover her error, until a fellow-servant detected the odor of camphor in her breath. In about two hours and a half she was seen by a medical man. She was then delirious, but gave rational answers when spoken to. She suffered no pain, but there was a feeling of giddiness: her face was pale, the pupils dilated, the countenance anxious, the hands and feet cold, the pulse 120 and feeble. Emetics were given, and in the evening the woman had recovered. (Med. Times, June 10, 1848, p. 88.) Mr. Hill, a pupil, communicated to me, in May, 1858, a case in which a lady, laboring under insanity, attempted suicide by taking camphor. She had at various times labored under obscure symptoms without any suspicion of the cause: but, it appears, that on one occasion she took sixty grains of camphor. She was seized with convulsions, frothing at the mouth, paleness of the face, and a scarcely perceptible pulse. In ten minutes she began to recover.

M. Raspail has advocated the use of camphor in large doses as a universal remedy for disease. This rash practice has been in some instances attended with dangerous effects. A man who had taken in divided doses about sixteen grains in twenty-four hours, complained of a sense of suffocation, difficulty of breathing, sickness, and great anxiety. (Jour. de Pharmacie, Fév. 1846, 121.) In the same journal three other cases are mentioned, in which alarming effects followed the injudicious use of this drug. The largest dose of camphor that has been taken, was in a case which occurred to Wendt, of Breslau. Eight scruples (160 grains) were swallowed by a drunkard, dissolved in spirit. The symptoms were giddiness, dimness of sight, delirium, and burning pain in the stomach. There was *no vomiting:* the man recovered! This case shows, that camphor cannot be regarded as an active poison. (Wibmer, op. cit. iii, 212.) In Orfila's experiments on animals, the mucous membrane of the stomach was found inflamed (ii, 493).

Treatment.—The free use of emetics and stimulants.

Analysis.—The camphor would probably be found in the state of lumps, or dissolved in spirit. No difficulty will occur in identifying this substance, when it has proved fatal and is found in the contents of the stomach. Its presence would be immediately known by its powerful and peculiar odor, which has been perceived throughout the whole body in dogs poisoned by it. If it were diffused in the form of lumps or powder, these might be easily separated from the contents, owing to the great insolubility of this substance. In general, it might be expected that some portions would float to the surface of water, in which it is very insoluble. In a doubtful case, the solid contents of the stomach may be concentrated and treated with a large quantity of alcohol: the alcoholic liquid filtered, and the camphor separated by adding water. It is a white solid,—possessing a well-known odor,—easily dissolved by alcohol, and again separated by water,—entirely volatile without residue, and burning with a rich yellow smoky flame. Camphor is soluble in chloroform, and this liquid may be used.as a means of separation from organic liquids.

CHAPTER XLI.

TOBACCO—SYMPTOMS AND APPEARANCES—CHRONIC POISONING—ANALYSIS—POISONING BY NICOTINA—PROPERTIES OF THE ALKALOID—COCCULUS INDICUS—SYMPTOMS AND EFFECTS—PICROTOXIA—DARNEL SEEDS—POISONING OF BREAD AND BEER—POISONOUS MUSHROOMS—HENBANE—SYMPTOMS AND APPEARANCES—HYOSCYAMIA—LETTUCE OPIUM—WOODY NIGHTSHADE.

TOBACCO.

Symptoms.—The leaves of the NICOTIANA TABACUM, variously prepared, either as tobacco or snuff, exert a powerful action on the body, although fatal cases of poisoning by tobacco are by no means common. The effects which this substance produces, when taken in a large dose, either in the form of powder or infusion, are well marked. The symptoms are faintness, nausea, vomiting, giddiness, delirium, loss of power in the limbs, general relaxation of the muscular system, trembling, complete prostration of strength, coldness of the surface with cold clammy perspiration, convulsive movements, paralysis, and death. In some cases there is purging, with violent pain in the abdomen; in others there is rather a sense of sinking or depression in the region of the heart, passing into syncope, or creating a sense of impending dissolution. With the above-mentioned symptoms there is dilatation of the pupils, dimness of sight, with confusion of ideas, a small, weak, and scarcely perceptible pulse, and difficulty of breathing. In Oct. 1855, a lunatic sailor swallowed from half an ounce to one ounce of crude tobacco, having, it is believed, kept it for some time in his mouth before he swallowed it. This was not known at the time of his admission into the asylum. After he had been placed in a warm bath he suddenly became insensible and motionless, the whole of the muscles were relaxed, the only indications of life being a feeble respiration, and a pulse scarcely perceptible. The pupils of the eyes were strongly contracted. In half an hour violent convulsions of a tetanic kind affected the limbs. There was profuse purging, and in the fluid evacuations some shreds of tobacco were found. This led to the knowledge that the patient had probably swallowed the poison. The stomach-pump was used with slight amendment,—the pupils became dilated: the symptoms however returned: there was vomiting as well as purging of mucus and blood with loud cries. The convulsions recurred with brief remissions, the limbs being at intervals rigidly flexed upon the body: there was grinding of the teeth. The pulse was feeble and rapid—scarcely perceptible: the action of the heart was very irregular. The pupils were again contracted and insensible to light. These symptoms continued until the patient died in a fatal syncope about seven hours after his admission. (Ed. Med. Jour. 1855–6, i, p. 643.) For three cases of poisoning by tobacco in which the persons recovered, see Reil, Jour. für Toxicol. 1857, 2 H. p. 568.

Tobacco acts *locally* as a poison: thus, when applied to wounded, abraded, or diseased surfaces, in the form of powder, juice, or as a decoction of the leaves, it may occasion the most alarming symptoms, and even death. (Orfila, ii, 404; also Pereira, Mat. Med. vol. ii, pt. i, p. 579.) This fact is of importance, as some quack remedies for skin diseases are composed of tobacco.

Appearances.—There have been but few instances in which the bodies of persons, poisoned by tobacco, have been inspected. In a case reported by Mr. Eade, a girl, æt. 18, injected as a clyster a decoction made by boiling *three drachms* of common shag tobacco in a pint of water. In half an hour she complained of faintness and feeling sick, and in another half hour she became quite collapsed, with cold sweats; she vomited, was slightly convulsed, and died in an hour and a half from the time at which she injected the clyster. On inspection, the heart was found very flaccid; there were three drachms of black fluid blood in the ventricles. The stomach contained food, but had no unusual appearance. The intestines presented no trace of inflammation or redness in any part, and there was no smell of tobacco (thirty-six hours after death) either in the intestines or in any part of the body. The head was not examined. (Med. Gaz. xliv, p. 823.) In the body of the lunatic whose case has been described (supra), the following appearances were met with forty hours after death. Cadaveric rigidity was very strongly developed. There was some congestion of the substance of the brain and the upper part of the spinal marrow (pons Varolii and medulla oblongata). The lungs presented no unusual appearance. The heart was empty, small, and contracted. In the abdomen the liver and kidneys were much congested. The mucous coat of the stomach presented several red patches. The intestines were contracted throughout and contained no fæcal matter. The mucous membrane was of a red color, partially abraded, and full; the intestines contained a mucous fluid tinged with blood. The mesenteric veins were distended with dark fluid blood. The bladder was contracted and empty. The blood was everywhere dark and liquid. (Ed. Med. Jour. 1855–6, i, p. 643.) Orfila found on examining the body of a dog killed by this substance, that the mucous membrane of the stomach was strongly reddened throughout.

Fatal dose.—Dr. McGregor has seen some of the most severe symptoms follow the administration of an injection which contained only *half a drachm* of tobacco in the form of decoction. (Lancet, Aug. 30, 1845, 240.) Dr. Paris witnessed a case, that proved rapidly fatal, in which a decoction of tobacco had been used as an injection in the attempted reduction of strangulated hernia (Med. Jur. ii, 418); and several instances of a similar kind are recorded by other writers. Dr. Pereira considers that it would not be safe to use more than fifteen or twenty grains under these circumstances, and he quotes an instance from Dr. Copland, in which death was caused by an infusion of *thirty grains*. (Mat. Med. vol. ii, pt. i, p. 579.) The fatal effects of tobacco may follow very speedily on its administration. Death has been known to take place in so short a period as three-quarters of an hour; and a case which occurred to M. Tavignot is reported to have terminated fatally in *eighteen minutes*. (Brit. and For. Med. Rev. No. xxiv, 562.)

Tobacco is rarely administered medicinally in substance. In a dose of five or six grains, *Snuff* acts as a powerful emetic, and in larger doses it produces symptoms of poisoning. It is a remarkable instance of the effect of habit, that the quantity thrust into the nostrils, as a sort of morbid luxury, does not appear to produce any directly noxious effects on the system. The diurnal allowance of many snuff-takers, introduced into the rectum in powder, would most probably give rise to serious symptoms, in one whose system was not habituated to the use of tobacco. The same may be observed of the practice of chewing and of *smoking* tobacco: in the latter case, the volatile oil of tobacco, as well as nicotina, are brought into immediate contact with the mucous membrane, producing faintness, giddiness, and sickness, in those not accustomed to the practice. In two instances, in which a large quantity of tobacco was consumed by smoking, death was the result. This involves a question as to the *chronic* form of poisoning by tobacco. Drs. Prout, Laycock, and Wright, consider that habitual smoking is injurious to health, because it is liable to disorder the digestive functions. This is denied by others, on account of the difficulty of showing that the health of inveterate smokers is damaged by the habit, or that their lives are shortened by it. Dr. Prout's view appears to me, notwithstanding, quite reasonable. A poisonous substance like tobacco, whether in powder, juice, or vapor, cannot be brought in contact with an absorbing surface like mucous membrane, without in many cases producing disorder of the system, which the consumer is probably quite ready to attribute to any other cause than that which would render it necessary for him to deprive himself of what he considers not merely a luxury, but an article actually necessary to his existence. The argument that cases cannot be adduced to show direct injury to health, proves too much, for the same difficulty exists in respect to the habit of opium-eating. (On this subject see Med. Gaz. xxxviii, 590, and Lancet, Aug. 1845, 240.) A controversy of still more recent date in reference to the effects of smoking has taken place in the medical journals, but nothing new has been elicited. It appears highly probable that in some instances nervous affections proceed from the inordinate use of tobacco in smoking. (Chemist, Jan. 1856, p. 246.)

Some doubt has existed whether the *vapor* of this substance, in tobacco manufactories, is or is not injurious to the health of the workmen employed. M. Parent-Duchâtelet considered that, after a time, it had no influence on health. The more recent researches of M. Melier have, however, led to the conclusion, that the vapors long respired are injurious. The primary effects are headache, nausea, languor, loss of appetite, and sleep; the secondary effects are manifested by a general disturbance of health. He attributes these symptoms to the nicotina which is volatilized. (Gaz. Med. Mai 3, 1845.)

Poisoning by tobacco has not often given rise to medico-legal discussion. This is the more remarkable, as it is an easily accessible poison, and the possession of it would not, as in the case of other substances, excite surprise or suspicion. In June, 1854, a man was charged with the death of an infant æt. 10 weeks by poisoning it with tobacco. He placed a quantity of tobacco in the mouth of the infant, with the view, as he stated, of making it sleep. The infant was completely narcotized and died on the second day. It is probably more extensively used to aid the purposes of robbers than is commonly believed; and there is reason to suppose that porter and other liquors sold in brothels, are sometimes drugged either with tobacco or snuff prepared from it. Scotch snuff is said to be used for this purpose. Dr. Ogston communicated a case of this kind to Dr. Christison (Op. cit. 850), in which tobacco was administered to a man in whiskey, and he soon afterwards died in a state of insensibility, without being able to give any account of the circumstances. Dr. Ogston detected *nicotina* in the contents of the stomach. An investigation took place in this metropolis, in the autumn of 1847, in which a man was charged with attempting to poison his wife, by administering to her snuff in ale. The woman's life was saved by the speedy use of the stomach-pump. The case was dismissed, as there was a want of clear proof of criminal intention. A question here arose as to what quantity of tobacco would destroy life. The medical witness is reported to have said, that a quarter of an ounce infused in a pint of liquid, would be sufficient to destroy three persons. This is no doubt true. Thirty grains have proved fatal, and twenty grains might even kill an adult. Many kinds of snuff are, however, extensively adulterated with various powders; some contain lime, and even red lead; hence they are not to be regarded as consisting of pure tobacco.

Analysis.—Tobacco may be found in substance in an organic liquid, or in the stomach; it may then be recognized by its odor, as well as by physical and botanical properties. Its poisonous effects are due to the presence of a peculiar volatile alkaloid, which, like conia, is liquid. It is called NICOTINA, and, accordinng to Dumas, it forms less than the 1-1000th part of tobacco. The late Dr. Pereira assigned a much larger proportion. According to him 1000 parts of Cuban tobacco yield 8·64 of nicotina, of Virginia, 10, and of smoking tobacco, 3·86. (Mat. Med. vol. ii, pt. i, p. 574.)

Nicotina.—This is the poisonous alkaloid of tobacco. It is a deadly poison, like prussic acid, destroying life in small doses and with great rapidity. It also resembles prussic acid in the fact that it is a compound of carbon, nitrogen, and hydrogen, and it contains no oxygen. I found that a rabbit was killed by a single drop in three minutes and a half. In fifteen seconds the animal lost all power of standing, was violently convulsed in its fore and hind legs, and its back was arched convulsively (opisthotonos). A frothy alkaline mucus escaped from its mouth, having the odor of nicotina (Guy's Hosp. Reports, Oct. 1858, p. 335). A case of poisoning by this alkaloid occurred in Belgium in 1851, and was the subject of a trial for murder. (Ann. d'Hyg. 1851, ii, pp. 147 and 167.) The Count and Countess Bocarmé were charged with the murder of the Countess's brother, a M. Fougnies, by administering to him nicotina while he was dining with them at the Château of Bitremont. The poison was forcibly administered. The deceased did not survive more than five minutes, and was not seen living by any of the attendants. The possession of the poison, as well as the moral evidence, fixed the crime on the Count, and he was condemned and executed. The appearances after death were to a great extent altered or destroyed by the pouring of some strong acid (acetic) into the mouth and over the body of the deceased, in order to conceal or remove the odor of nicotina. M. Stas detected the poison in small quantity in the tongue, throat, stomach, liver, and lungs of the deceased, as well as in a wooden plank of the floor near to which he was sitting. A second case of poisoning by this alkaloid, and the only case recorded in this country, occurred in London as an act of suicide in June, 1858. A gentleman swallowed a quantity of nicotina from a bottle, and almost immediately afterwards was seen in the act of falling to the floor. He was carried to an adjoining room, but before this could be reached, he was dead. The symptoms noticed were that the deceased stared wildly: there were no convulsions, and he died quietly, heaving a deep sigh in expiring. In producing these effects nicotina resembles prussic acid. The quantity of nicotina taken, could not be determined. The deceased appears to have been rendered immediately insensible, and to have died in from three to five minutes, after having taken the poison.

The *appearances* observed were a general relaxation of the muscles, prominent and staring eyes, bloated features, great fulness, with lividity about the neck. There was no odor resembling nicotina or tobacco perceptible about the body. When examined, between two and three days after death, putrefaction had occurred, especially in the course of the veins. The swelling of the neck was found to arise from an effusion of dark liquid blood. The scalp, as well as the membranes of the brain were filled with dark-colored blood. The lungs were engorged and of a dark purple color. The cavities of the heart were empty, with the exception of the left auricle, which contained two drachms of dark-colored blood. The stomach contained a chocolate-colored fluid (reserved for analysis): the mucous membrane was of a dark crimson-red color from the most intense congestion. There was no odor excepting that of putrefaction. The liver was also congested and of a purple-black color. The blood throughout the body was black and liquid: but in some parts it had the consistency of treacle. I found nicotina in small quantity in the contents of the stomach, also in the liver and lungs: but as these organs had been placed in contact with the stomach, it could not be inferred that the poison had been absorbed and deposited in them.

Analysis.—A sample of nicotina which I examined had a pale amber color, and evolved a peculiar acrid odor, affecting the nose and eyes, resembling stale tobacco smoke. It had the consistency of a thin oil, gave a greasy stain to paper, which soon disappeared owing to its volatility. When heated on platina or on paper it burned with a bright yellow flame, emitting a thick black smoke. It was powerfully alkaline, and imparted a strong alkaline reaction to water. The aqueous solution, even when much diluted, retained the peculiar odor. Nicotina is dissolved by alcohol and ether, and the latter liquid will remove it from its aqueous solution. 1. Chloride of platina produces in the aqueous solution an orange yellow crystalline precipitate. 2. Corrosive sublimate gives a white precipitate. 3. Arsenio-nitrate of silver gives a yellow precipitate. In all these characters nicotina resembles ammonia: the differences, apart from the odor, which is an important distinction, are: 4. Iodine water gives a brown precipitate (in ammonia there is no precipitate, the color is discharged). 5. Tannic acid gives a whitish-yellow precipitate (in ammonia there is no precipitate, but a red color is imparted). 6. Gallic acid gives no precipitate (in ammonia it produces a pinkish red color, rapidly changing to an olive green). 7. Sulphuric acid and bichromate of potash produce a green color by the liberation of oxide of chromium. (See Guy's Hosp. Reports, Oct. 1858, p. 354.)

Organic mixtures.—To separate nicotina from the contents of the stomach, these should be digested in cold distilled water, acidulated with sulphuric acid in the proportion of a drop to an ounce. This liquid is strained, filtered, and the residue pressed. It is then to be evaporated to one-half in a water-bath—digested with its bulk of cold alcohol, filtered, and the alcoholic liquid evaporated in a water-bath. The sulphate of nicotina is now dissolved out of the residue by a small quantity of water: the solution is rendered alkaline by potash and then shaken in a tube with its bulk of ether; the ethereal liquid is allowed to evaporate in a series of watch-glasses, and if nicotina be present the alkaloid will be left in small oily-looking globules. The odor may not be perceptible until the residue is heated, when its peculiar acridity will be brought out. A few drops of water should be added to the residue in each glass: it will then be found to be strongly alkaline: and the different tests may be applied. It was by this process that I discovered the poison in the body of the gentleman whose case is above related. In reference to the rabbit killed by a single drop (supra), nicotina was found in the stomach and its contents: there was a trace found in half an ounce of the blood of the animal, and the poison was clearly detected after a week in the tongue and soft parts of the throat of the animal, but there was no trace of nicotina in the liver, heart, or lungs.

COCCULUS INDICUS.

Symptoms and effects.—This is the fruit or berry of the MENISPERMUM COCCULUS (*Levant Nut*), imported from the East Indies. It contains from one to two per cent. of a poisonous alkaloid (*Picrotoxia*). The seeds, in powder or decoction, give rise to nausea, vomiting, and griping pains, followed by stupor and intoxication. There are, so far as I am aware, only two well-authenticated instances of this substance having proved fatal to man. Several men suffered from this poison in 1829, near Liverpool: each had a glass of rum strongly impregnated with Cocculus Indicus. One died that evening; the rest recovered. (Traill's Outlines, 146.) Of the second case, the following details have been published. A boy, æt. 12, was persuaded by his companions to swallow two scruples of the composition used for poisoning fish. It contained Cocculus Indicus. In a few minutes he perceived an unpleasant taste, with burning pain in the gullet and stomach, not relieved by frequent vomiting,—as well as pain extending over the whole of the abdomen. In spite of treatment, a violent attack of gastro-enteritis supervened, and there was much febrile excitement, followed by delirium and purging, under which the patient sank on the nineteenth day after taking the poison. On inspection, the vessels of the pia mater were found filled with dark-colored liquid blood. There was serous effusion in the ventricles of the brain, and the right lung was congested. In the abdomen there were all the marks of peritonitis in an advanced stage. The stomach was discolored, and its coats were thinner and softer than natural. (Canstatt, Jahresbericht, 1844, v, 298.)

London porter and ale are considered, and in some instances with propriety, to owe their intoxicating properties to a decoction, or extract of these berries. The fraud is perpetrated by a low class of publicans. They reduce the strength of the beer by water and salt, and then give to it an intoxicating property by means of this poisonous extract. A medical man consulted me some years since, in reference to the similarity of cerebral symptoms suffered by several of his patients in a district in London. It was ascertained that they were supplied with porter by retail from the same house. The effects produced are remarkable: there is a strong disposition to sleep, and at the same time wakefulness. There is lethargic stupor, with a consciousness of passing events, but a complete loss of voluntary power. It is a kind of nightmare feeling, altogether different from healthy sleep. Cocculus indicus is sometimes used by robbers to intoxicate their victims, and to this form of intoxication, the term "hocussing" is applied. This substance is applied to no useful purpose whatever, either in medicine or the arts; and, under a proper system of medical police, its importation would be strictly prohibited.

Cocculus indicus is sometimes used for the purpose of taking fish. As it destroys them by poisoning them, it is a question whether the fish thus killed, can be safely eaten as food. M. Chevalier has examined this subject; and the conclusion to which he has come is, that, although instances may be cited of the fish being used with impunity as food, yet it must be regarded as a dangerous practice. The result depends on the quantity of poison used: thus, when from ten to fifteen grains of the berries were employed, and the fish was afterwards given to animals, the noxious effects were as strongly marked as if they had swallowed the poison. (Ann. d'Hyg. 1843, i, 343.)

The shell appears to act like an emetic, while the kernel, which alone contains picrotoxia, is the seat of the poison. On this ground it has been contended that the entire seed or fruit is not legally a poison (see ante, p. 27.)

From the facts collected by Wibmer (Arzneimittel. *Menispermum*), neither cocculus indicus nor picrotoxia, which forms only 1-100th part of the kernel, appears to be possessed of very active properties upon large animals. Orfila gave 3·38 grains of picrotoxia to a good-sized dog: it produced frequent vomiting, but the animal soon recovered.

one-tenth part with rye, the persons partaking of the bread suffered from giddiness, headache, nausea, vomiting, deafness and cramps. (Medical Gazette, xlv, 872; Ann. d'Hyg. 1853, ii, p. 147.) Among the symptoms in other cases there has been noticed a sense of burning heat in the mouth and throat, with confusion in the head, trembling, and a small irregular pulse. (See Ed. Monthly Jour. Aug. 1850, p. 180.) When these symptoms attack simultaneously many persons who partake of the same bread there is strong ground for suspicion.

A similar accident occurred in Germany, the seeds of the darnel having become mixed with those of barley and made into bread. The prominent symptom was giddiness in a severe form. As a result of this mixture the poisonous principle of darnel (which is capable of resisting a baking heat) may find its way into beer or brandy. (Casper's Vierteljahrschrift, Oct. 1857, p. 343.) A wet season is favorable to the growth of darnel with the varieties of corn. The seeds are difficult of separation.

Rye-bread is not much used in this country, but the accidental presence of the *ergot* may sometimes account for the symptoms of poisoning which have been observed. (See Ann. d'Hyg. 1834, ii, 179; 1835, ii, 240; 1843, i, 41, 347; Henke, Zeitschrift der S. A. 1842, ii, 185; 1844, i, 286, ii, 215.)

Analysis.—The botanical characters of the plant. Pfaff has lately examined darnel, in order to discover a poisonous alkaloid; but there was no trace of such a substance. By distillation with water he obtained two kinds of ethereal oil, one lighter and the other heavier than water; they were colorless, and had the odor of fusel-oil.

MUSHROOMS—(FUNGI.)

Cases of poisoning by mushrooms (FUNGI) are by no means unusual as the result of accident. According to the late Dr. Badham there are five thousand recognized species, of which only a few can be safely eaten. Among them the *Agaricus campestris* and *esculentus* are perhaps most commonly employed as articles of food. It is a curious fact, that the poisonous properties of mushrooms vary with climate, and probably with the season of the year at which they are gathered. Another circumstance deserving of notice, is, that by idiosyncrasy, some individuals are liable to be seriously affected even by those species which are usually regarded as innocent. Some species which are poisonous in this country, are used freely by the Russians: it appears they are in the habit of salting, boiling, and compressing them before they are eaten; and this may in some instances suffice to account for their having no noxious effects. Dr. Badham states that the *Agaricus campestris* or common mushroom, which is largely eaten in England, is regarded as poisonous in Rome, and is accordingly rejected; while some varieties, which in this country would produce symptoms of poisoning, are eaten in Italy with impunity. There do not appear to be any satisfactory rules for distinguishing the mushrooms which are wholesome from those which are poisonous. The best test is that assigned by Dr. Christison—namely, that the poisonous vegetable has an astringent styptic taste; as well as a disagreeable, but certainly a pungent odor. All mushrooms that are highly colored, or grow in dark and shady places, are generally poisonous.

Symptoms and Effects.—The noxious species of mushrooms act sometimes as narcotics, at others as irritants. It would appear from the reports of several cases, that when the narcotic symptoms are excited, they come on soon after the meal at which the mushrooms have been eaten, and that they are chiefly manifested by giddiness, dimness of sight, and debility. The person appears as if intoxicated, and there are singular illusions of sense. Spasms and convulsions have been occasionally witnessed among the symptoms when the case has proved fatal. Dr. Peddie has related three instances of poisoning by mushrooms (Edin. M. and S. J. xlix, 200), in which the poison acted as a pure narcotic; there was no pain in the abdomen, nor irritation in the alimentary canal. The narcotic symptoms began in half an hour with giddiness and stupor; the first effect with one patient was, that every object appeared to him to be of a blue color. The three patients recovered, two of them rapidly. When the drowsiness passes off, there is generally nausea and vomiting; but sometimes vomiting and purging precede the stupor. If the symptoms do not occur until many hours after the meal, they partake more of the character of irritation,—indicated by pain and swelling of the abdomen, vomiting, and purging. Several cases, in which the symptoms did not appear until after the lapse of fourteen hours, are reported in the Medical Gazette (vol. xxv, p. 110). In some instances the symptoms of poisoning have not commenced until thirty hours after the meal; and in these, narcotism followed the symptoms of irritation. It might be supposed that these variable effects were due to different properties in the mushrooms; but the same fungi have acted on members of the same family, in one case like irritants, and in another like narcotics. In most cases recovery takes place, especially if vomiting be early induced. In the few instances which have proved fatal, there has been greater or less inflammation in the stomach and bowels, with congestion of the vessels of the brain. (See Med. Gaz. vol. xlvi, p. 307, vol. xlvii, p. 673; and Journ. de Chimie Méd. 1853, p. 694.) Balardini states, that of sixty-eight cases of poisoning by mushrooms, which occurred in the province of Brescia, during a period of twenty years, twenty proved fatal. The principal symptoms were nausea, uneasiness in the abdomen, giddiness; a state resembling intoxication; vomiting and purging; loss of power of locomotion, with convulsions. (Canstatt's Jahresbericht, 1844, v, 300.) In some persons even the edible mushrooms will procuce disorder of the stomach and bowels as a result of idiosyncrasy. In six cases which occurrred to Dr. Keber, in which the *Helvella esculenta* had caused symptoms of poisoning, the patients were jaundiced as soon as the vomiting had ceased. The principal symptom was urgent vomiting: but one girl, æt. 18, fell into a state of coma, from which she did not recover for three days. It was probable that, in this instance, the noxious effects were due to season. (Gaz. des Hôp. Oct. 10, 1846.) The common truffle (*Morchella esculenta*) has been known to give rise to severe symptoms of irritant poisoning. In some reported cases (Ed. Med. and Surg. Journ. Oct. 1845, 530; and Ann. d'Hyg. 1845, vol. i, p. 214), it is probable that the truffles had undergone decomposition before they were eaten.

Ketchup, a liquor made from mushrooms, has occasioned faintness, nausea, and severe pain in the abdomen, disappearing only after some hours. (Dub. Med. Press, Sept. 24, 1845, 195.) There are two modes of explaining this effect: 1, either that the person labors under an idiosyncrasy with respect to mushrooms in general; or 2, that noxious have been gathered by mistake for esculent mushrooms. A case is on record which shows that a medical jurist may be easily misled when any active poison is mixed with and administered in a dish of mushrooms. A servant girl poisoned her mistress, by mixing arsenic with mushrooms. This person died in twenty hours, after suffering severely from vomiting and colicky pains. On dissection, the stomach and intestines were found inflamed. Death was ascribed to the effects of the mushrooms, which were considered to have been unwholesome; and the fact of poisoning only came out many years afterwards, by the confession of the prisoner! This shows with what a watchful eye such cases should be examined; in the absence of an analysis of the contents of the stomach, it would be impossible to develope the truth.

In the Lancet of June 28, 1856, p. 716, a case is related in which some poisonous fungi accelerated death, if they did not actually cause it. The man had eaten stewed mushrooms and died rather suddenly, having shortly before complained of pain in his bowels. The mucous membrane of the gullet, as well as that of the stomach, was inflamed.

Treatment.—The free use of emetics and castor oil.

Analysis.—The discovery of portions of the fungus in the matter vomited, or the description of the food eaten, will commonly lead to a recognition of this form of poisoning. The poisonous principle contained in mushrooms is called *Fungin:* it appears to be of a volatile nature, and soluble in hot water; for, some varieties of noxious mushrooms may be eaten with impunity, when they have been well boiled in water and afterwards pressed. One of the most poisonous in this country, *Amanita muscaria*, or Fly-mushroom, renders the water in which it is boiled so poisonous, that animals are killed by it, while the boiled fungus itself has no effect upon them. The liquid procured from it is used as a fly-poison, whence the name of the mushroom is derived. It is an autumnal fungus known by its rich orange-red color.

HENBANE—(HYOSCYAMUS NIGER.)

All the parts of this plant are poisonous. The seeds produce the most powerful effects, then the roots, and lastly, the leaves. The vapor evolved from the fresh-cut leaves has been known to produce giddiness, stupor, and syncope. In small or medicinal doses, henbane has a narcotic action; but when taken in large doses, it produces effects on the spinal marrow as well as on the brain.

Symptoms.—The best summary of these is given by Wibmer (Arzneimittel. Art. HYOSCYAMUS NIGER). When the dose is not sufficient to destroy life, the symptoms are,—general excitement, fulness of the pulse, flushing of the face, weight in the head, giddiness, loss of power and tremulous motion of the limbs, somnolency, dilatation of the pupils, double vision, nausea and vomiting. After a time these symptoms pass off, leaving the patient merely languid. When a large quantity of the *root* or *leaves* has been eaten, an accident which has occurred from the plant having been mistaken for other vegetables, more serious effects are manifested. In addition to the above symptoms in an aggravated form, there will be loss or incoherency of speech, delirium, confusion of thought, insensibility, coma, and, sometimes, a state resembling insanity; the pupils are dilated, and insensible to light, there is coldness of the surface, cold perspiration, loss of power in the legs, alternating with tetanic rigidity and convulsive movements of the muscles, the pulse small, frequent and irregular, the respiration deep and laborious. (See Med. Gaz. vol. xlvii, p. 640.) Occasionally there is nausea, with vomiting and purging. Death takes place in a few hours or days, according to the severity of the symptoms. The special effect of this poisonous plant is manifested in its tendency to produce a general paralysis of the nervous system. As an instance of the singular train of symptoms occasionally produced by it, Dr. Houlton states, that in a monastery where the roots had been eaten for supper by mistake, the monks who partook of them were seized in the night with the most wonderful hallucinations, so that the place became like a lunatic asylum. One monk rang the bell for matins at twelve o'clock at night: of those of the fraternity who attended to the summons, some could not read, some read what was not in the book, and some saw the letters running about the page like so many ants! (Lancet, July 6, 1844, p. 479.)

Among the reported cases of poisoning by henbane is the following: A woman collected in a field a quantity of the root by mistake for parsnips. They were boiled in soup, of which nine persons in the family partook without remarking any particular taste. Very shortly afterwards, the whole of these persons felt uneasy, and complained of a bitter acrid taste in the mouth, with nausea. The pupils of the eyes were dilated, and there was indistinctness of vision. These symptoms were followed by great restlessness, convulsions, and continued delirium. The patients successively lost the power of vision, hearing, and voice, and were affected with stupor and insurmountable somnolency. (Ed. Med. and S. J. Oct. 1844, p. 562.) Orfila relates the cases of two men who ate the young shoots of the plant. The first effect was, that the earth seemed to pass suddenly from under them: the tongue became paralyzed, and their limbs were cold, torpid, paralyzed, and insensible; the arms were in a state of spasmodic action, the pupils were dilated, the look was fixed and vacant, breathing difficult, the pulse small and intermittent. Besides these symptoms, there was the spasmodic grin (*risus sardonicus*) with delirium; and the jaws were spasmodically closed. Under treatment, the men recovered in the course of two days. (Op. cit. 4ème ed. ii. 264.)

When the extract or decoction is introduced into the rectum, or applied externally to a wound, similar effects are observed to follow. In a case quoted by Orfila, in which a decoction of the plant was used as an injection, the patient suffered from all the symptoms of apoplexy, with the exception of the absence of stertorous breathing.

Appearances.—One fatal case of poisoning by the roots of henbane is quoted by Orfila, and another by the leaves is reported by Wibmer (op. cit. p. 147). The appearances consisted in a general congestion of dark-colored liquid blood in the venous system. The lungs and brain especially manifested this condition. There are commonly no marks of irritation or inflammation in the stomach and bowels.

Fatal dose.—There are no data by which we can determine the relative activity of henbane. In powder the medicinal dose of the leaves is from five to ten grains; of the seeds, from three to eight grains. The dose of the tincture is from half a drachm to two drachms, and of the extract, from five to ten grains; but this preparation is more likely to vary in strength than any of the others. Dr. Burder states, that he has observed great inconvenience to follow from a dose of ten minims of the tincture repeated every six hours. After three or four doses there was pain, with oppression of the head. Ten minims given in doses at an interval of six hours, were followed by pain in the head, flashing of light before the eyes, and delirium. (Lancet, July 6, 1844, 480.) There may be, as in the case of opium, an idiosyncrasy with respect to this drug. Twenty *seeds* have produced complete delirium (Wibmer, op. cit. 147), and the same writer states, that, in one instance, alarming symptoms were caused by seven grains of the extract (154). The poisonous properties of the plant are affected by soil and season. They are most developed in it while the seeds are being formed.

Treatment.—The speedy expulsion of the poison by emetics and castor oil.

Analysis.—When the vegetable has been eaten, it can be identified only by its botanical characters. The seeds are very small and hard, they are honeycombed on the surface, and may be easily confounded with those of belladonna. The poisonous properties of henbane are known to be owing to a crystalline alkaloidal body, which is called *Hyoscyamia.* It is very difficult of extraction. The crystals have a silky lustre,—they are not very soluble in water, but are easily dissolved by alcohol and ether. It has an alkaline reaction, and its saline solutions are precipitated by tannic acid. It has an acrid disagreeable taste, resembling that of tobacco. It is highly poisonous, and causes dilatation of the pupils.

Professor Schroff, of Vienna, has performed some experiments on himself and a friend with small doses of this alkaloid. The symptoms produced were giddiness, unsteadiness of gait, with great dryness in the mouth and throat, so that nothing could be swallowed; there was headache, with impairment of the senses of tasting and smelling, and after a time a strong tendency to sleep. The pupils were dilated. With regard to this symptom it was observed that by local application, a solution of hyoscyamia produced intense and continued dilatation. The medicinal dose is assigned by him at from one-sixtieth to one-twentieth of a grain. Its poisonous operation begins to be manifested with one-tenth of a grain. (Wochenblatt, June 16, 1856; Brit. and For. Med. Rev. vol. xix, Jan. to April, 1857, p. 260. See also Reil, Journal für Toxicologie, 1857, 2 H. p. 277.)

LACTUCARIUM—(LACTUCA.)

Symptoms and Effects.—The two species of lettuce, known under the names of LACTUCA SATIVA and VIROSA, contain a principle which is possessed of feebly narcotic properties. Orfila has found that the extract prepared by evaporation at a low temperature, acts upon the brain and nervous system of animals; although very large doses were required for the production of narcotic effects. There is no record of these plants having exerted a poisonous action in the human subject.

The inspissated juice of the lettuce, is well known under the name of *lactucarium* or *lettuce-opium.* (See Pereira, Mat. Med. vol. ii, pt. ii, p. 36.) The Lactuca Virosa yields three times as much as the Lactuca Sativa; and half a grain of it, according to Dr. Fisher, is equivalent to two or three grains of that obtained from the Lactuca Sativa. (Med. Gaz. xxv, 862.) The juice, when it first escapes, is of a milky white hue, but, in drying, it forms an extract in small irregular dry masses of a brown color, a bitter taste, and with an odor similar to that of opium. It has a weak narcotic action when given in doses of from five to twenty grains. It varies much in strength. Wibmer found that *two grains* caused headache and somnolency. (Op. cit. 200.)

Analysis.—By the smell only, it may be mistaken for opium. It is but little soluble in water, and after long boiling, it forms a brown turbid solution which produces a greenish tint with a persalt of iron. It therefore contains no meconic acid. On examining a good specimen I have not found any trace of morphia. This shows that the *odor* of opium may exist in substances which do not contain meconate of morphia. Nitric acid gives a yellowish tinge to the decoction, as it does to most other vegetable solutions. It is bitter to the taste, which appears to be owing to the presence of a bitter principle called *lactucin,* upon which its feebly narcotic properties probably depend. There are no tests for lactucarium, further than the color, the opiate odor, with the want of solubility, and the absence of the other chemical characters of opium. In the plant it is combined with malic acid, potash, and resin. (Fisher, loc. cit.)

NIGHTSHADE—(SOLANUM.)

Symptoms and Effects.—There are two species of this plant—the *Solanum Dulcamara, Bitter-sweet* or *Woody-nightshade,* which has a purple flower and bears red berries; and the *Solanum Nigrum,* or *Garden-nightshade,* with a white flower and black berries. Dunal gave to dogs four ounces of the aqueous extract, and, in another experiment, 180 ripe berries of the Dulcamara, without any ill effects resulting. On the other hand, Floyer states that thirty of the berries killed a dog in three hours. (Wibmer, op. cit. SOLANUM.) These differences may perhaps be reconciled by supposing that the active principle *Solania,* on which the poisonous properties of both species depend, varies in proportion at different seasons of the year. In one instance a decoction of the plant is said to have produced in a man dimness of sight, giddiness, and trembling of the limbs—symptoms which soon disappeared under slight treatment. (For a case of poisoning, by the decoction, see Med. Gaz. vol. xlvi, p. 548.) Orfila found that the extract of solanum nigrum had a feeble effect as a poison: and the fatal cases reported to have been caused by it, are perhaps properly referable to belladonna (*Deadly-nightshade*), for which it may have been mistaken. The single death from Dulcamara reported in the Registration returns for 1840, may have been due to a mistake of this kind.

Nevertheless the berries of the solanum nigrum, in one instance at least, produced serious effects in three children who had eaten them. They complained of headache, giddiness, sickness, colic, and tenesmus. There was copious vomiting of a greenish-colored matter, with thirst, dilated pupils, stertorous respiration, convulsions, and tetanic stiffness of the limbs. One child died in the acute stage: the others died apparently from secondary consequences during treatment. (Orfila, op. cit. 4ème ed. ii, 273.) From three to four berries of this plant have been found to produce sleep. In September, 1853, the red berries of the *Woody-nightshade* are stated to have caused the death of a boy, æt. 4, under the following circumstances. He had eaten some of the berries, and at first did not appear to suffer from them; but eleven hours afterwards he was attacked with vomiting, purging, and convulsions, which continued throughout the day; the child being insensible in the intervals. He died convulsed in about twenty-four hours. The vomited matters were of a dark greenish color and of a bilious character. Other children had partaken of the berries at the same time: but one of them suffered only slightly. (Lancet, June 28, 1856, p. 715.)

Solania. Analysis.—The plants can only be identified botanically by an examination of the leaves and berries. The active principle in both is an alkaloid, *Solania*, which is itself a poison, although not very energetic. Two grains of the sulphate of solania killed a rabbit in a few hours. The action of solania and its salts upon animals has been investigated by Dr. J. Clarus, of Leipzig (Reil, Journal für Toxicologie, 1857, 2 H. p. 245). According to him, solania exalts the sensibility of the skin like strychnia, but destroys life by producing paralysis of the muscles of the chest, like conia or nicotina. It appears to occupy an intermediate place between nicotina and strychnia. It differs from atropia, daturia and hyoscyamia in not producing stupor or delirium, dilatation of the pupils, or paralysis of the sphincter muscles. He regards it as a narcotico-acrid (cerebro-spinal) poison, and assigns the medicinal dose of the acetate for an adult at one-sixth of a grain. *Tests*—1. Chromic acid gives a sky-blue color passing to a green. 2. Concentrated sulphuric acid produces an orange-red colour passing through a yellow to a violet. 3. Nitric acid with the vapor of ammonia produces a rose-red color. 4. The solution is not precipitated by chloride of platina or iodide of potassium.

SPINAL POISONS.

CHAPTER XLII.

POISONING BY NUX VOMICA—SYMPTOMS—APPEARANCES—FATAL DOSE AND PERIOD OF DEATH—ANALYSIS—ST. IGNATIUS'S BEANS—UPAS TIEUTE—JAVA AND MADAGASCAR POISONS—CURARA—CURARINA—STRYCHNIA AND ITS SALTS—SYMPTOMS—APPEARANCES—FATAL DOSE AND PERIOD OF DEATH.

***Remarks.*—The poisons belonging to this section are so named from the fact that their chief action is exerted on the spinal marrow, the brain being unaffected. With an exaltation of sensibility there are the most violent convulsions, in which the muscles become rigid and fixed, producing a state of the body resembling tetanus. There is no stupor or delirium; consciousness is generally retained until just before death. *Strychnia* is the poisonous alkaloid which produces these remarkable effects. It was discovered in 1818, in the seed of nux vomica, by Pelletier and Caventou. It is peculiar to plants or seeds grown in tropical climates, and has hitherto been found in five only.**

NUX VOMICA.

This is the seed of the *Strychnos Nux Vomica*, which is a native of Coromandel, Ceylon, and the jungles of Bengal. The seed has the shape of a flat round kernel, of a grayish-brown color, of about the size of a shilling, but much thicker. It is covered with a fine silky fibrous down, radiating from the centre. The fruit of the tree which yields it is said to be of the size of a pear, and to contain from three to five of these seeds in the midst of a pulp. As in the case of the cherry-laurel (p. 620), the pulp is described as not being poisonous, while the seeds contain *strychnia*, one of the most deadly poisons known. One seed weighs about thirty grains in the dry state. The proportion of strychnia contained in the seeds has not been accurately determined. Gmelin has assigned the proportion at (0·4) less than one-half per cent. (Chimie Organique, p. 111), but Mr. Horsley considers it to amount to one per cent. by weight. The strychnia is combined with a vegetable acid, the strychnic or igasuric acid, and this renders the alkaloid soluble in water. There is another poisonous alkaloid associated with strychnia in the seed, namely, *brucia*. This acts upon the body like strychnia, but with about only one-sixth of the power. In addition to these poisonous alkaloids, the seed contains woody fibre, gum, wax, and oil. Nux vomica is commonly sold to the public in the form of a grayish-brown powder, at the rate of eight pence an ounce. In this state it may be mistaken for the powders of numerous medicines—ipecacuanha, bark, &c.; but it is known by its intensely bitter taste, which is persistent, and by the fact that it strikes a deep orange-red color when treated with strong nitric acid. Most medicinal powders give, with strong nitric acid, a dingy green or brown color. Nux vomica powder may, however, in the process of grinding, be mixed with other innocent powders, which will entirely destroy this chemical reaction. In the case of Reg. v. Wren (Winchester Lent Assizes, 1851), I found the nux vomica, used by the prisoner in an attempt at poisoning, to be strongly impregnated with the powder of guaiacum, so that nitric acid produced with it a deep green color. This fact served to identify the sample, and it was proved that it had derived the impregnation from its having been ground in a mill in which guaiacum had been previously ground. In other cases the powder may be mixed with farinaceous substances. Nux vomica, if used as poison at all, is employed in the form of a powder. Its chief use is for the destruction of vermin. It has, however, occasioned numerous deaths, chiefly as a result of suicide. Owing to its bitter taste, its presence in an article of food would be detected in any attempt at murder. In the case of Wren (supra), the poison was mixed in milk by the prisoner, an ignorant lad; but it completely altered the color and taste of the milk, and thus led to detection.

Symptoms.—At a period, varying from a quarter of an hour to an hour or longer, after the poison has been swallowed, the patient suddenly loses the power of walking, standing, or moving; he is then seized with twitchings of the muscles, followed by shocks or tetanic spasms, affecting the whole of the muscular system—the body becoming rigid, the limbs stretched out, and the jaws so fixed, that considerable difficulty is experienced in introducing anything into the mouth. The muscles of the face are fixed by spasmodic contraction producing the sardonic grin, and the body sometimes assumes the state of opisthotonos: there is consciousness, and the intellect is generally clear. This spasmodic state ceases for a time, but after a short interval reappears; and the chest may become so fixed as to give the feeling of impending suffocation. After repeated attacks, generally increasing in severity, the patient dies, either suffocated or exhausted. Drowsiness, giddiness, and a feeling of general illness have in some instances preceded the spasms; vomiting, pain in the abdomen, and other symptoms of irritation, have also been occasionally witnessed among the symptoms. A woman, æt. 23, swallowed 120 grains of powdered nux vomica, mixed with water. Sometime after she was suddenly seized with giddiness, a loss of power in her legs, and a general feeling of stiffness in the body, especially in the neck. She fell, was carried home, and was then seen by a medical man. He found her with her face flushed, pupils dilated, the pulse quick, as well as the breathing, numbness and stiffness of the legs, and a feeling of constriction across the chest. Every two or three minutes there was a convulsive tetanic spasm throughout the body, but it was only momentary, like an electric shock. The stomach-pump was used and other remedies were applied, under which she recovered, suffering only from a slight stiffness of the jaws and debility. (Lancet, Dec. 15, 1849.)

In another case, in which a similar dose was taken by a man, the first symptoms were profuse perspiration, with twitchings of the muscles, ending in a strong and general tetanic spasm. The mind was clear, and questions were answered rationally during the intervals of the spasms.

It was observed that these were brought on when any attempt was made to wipe the perspiration from his face. (Lancet, July 5, 1856, p. 11.) Two hours after he had taken the poison, an emetic of sulphate of zinc was given to him, and it produced violent vomiting. The tetanic spasms then gradually subsided. A man, æt. 20, swallowed 90 grains of the powder. Spasms of the muscles appeared in ten minutes. In three-quarters of an hour he was in a profuse perspiration, the skin of the head and face congested, the eyes suffused, the pupils slightly contracted, and the pulse hard. Fits of tetanus, each lasting about half a minute, then attacked him. All the muscles were rigid, and his breathing appeared for the time suspended. The muscles were then relaxed, and he was able to answer questions. In two days he recovered. (Lancet, Oct. 22, 1853, and Med. Chir. Rev. Jan. 1854, p. 292.) The spasm does not always involve the chest. A youth, æt. 19, swallowed a tea-spoonful (= 65 grains) of the powder of nux vomica in a cup of cocoa; in fifteen minutes he was attacked with convulsions, and fell from his seat. The whole of the body was affected with convulsive twitchings, but these were unattended with pain. The chest was not affected, and there was no difficulty of breathing. He felt their approach, but could not describe the sensation. The stomach-pump was used with benefit, and the symptoms disappeared in three hours. (Med. Times and Gaz. April 28, 1855, p. 424.)

A physician took by mistake five grains of the alcoholic extract of nux vomica in two pills, and his wife took a similar dose at the same time. They had tea, and felt no ill effects for forty minutes, when the physician, in rising to go to the door, suddenly exclaimed "Hold me!" The wife rose to render assistance, but she was suddenly fixed in her position by muscular spasm. In ten minutes they were seen by a medical man, who prescribed emetics, which acted speedily and powerfully. The two patients were fixed by spasm to the chairs on which they were sitting, the convulsions coming on at intervals, and being rapidly succeeded by a relaxation of the muscles. During the fit, the heads were drawn backwards, there was a spasmodic clenching of the teeth, the heels were fixed to the ground, the eyes protruding from their sockets, and both exclaimed, "Hold me! hold me!" although there was a person on either side of each. In about five hours, under the use of emetics, the spasms subsided. On the next day they recovered, but suffered from some debility. (Med. Times and Gaz. Jan. 16, 1858, p. 79.) The subject of poisoning by nux vomica, including a collection of cases, has been ably treated by Dr. Husemann, in Reil's Journal für Toxicologie, 1857, 2 H. p. 469.

Chronic poisoning.—Medicinal doses frequently repeated may produce all the effects of chronic poisoning. A lady took three grains of the powder of nux vomica thrice daily, for sixteen days (=144 grains in the whole). There were no obvious effects for a fortnight, when there was purging with colicky pains. The nux vomica was withdrawn, and on the fifth day after its withdrawal, the patient suffered from ringing in the ears, drowsiness, increased sensibility to light and sound, numbness and impairment of speech. On the ninth day she lost her speech, tetanic symptoms, with twitchings of the muscles of the face and arms set in, as well as fixation of the jaws (trismus). There were slight intervals of relaxation, during which she swallowed with difficulty. The pupils were dilated, and the skin was hot. The spasms increased, and on the twelfth day the breathing became affected. In the evening of this day, she was seized with a strong tetanic convulsion, in which respiration ceased, the face became livid, the brows contracted, the lips were drawn widely apart, and the features greatly distorted, assuming the sardonic grin. During the night she had four similar paroxysms, and died apparently exhausted on the twenty-eighth day after she had commenced with the nux vomica, and on the twelfth day after its discontinuance. (Lancet, June 14, 1856, p. 654.)

Appearances.—These are not very characteristic. There is congestion of the brain and its membranes, with engorgement of the lungs. The heart has been found, in some instances, empty and flaccid (see Med. Times and Gaz. 1856, Feb. 9, p. 149): while in others it was distended with dark-colored and fluid blood. The mucous membrane of the stomach and intestines is occasionally congested. The powder has been found adhering tenaciously to this membrane. The spasmodic state of the muscles has continued for some time after death. In the fatal case of chronic poisoning above referred to, there was congestion of the membranes of the brain, and the heart was contracted and empty. There was a slight inflammatory appearance in the ileum. (Lancet, June 14, 1856, p. 654.)

Fatal dose. Period of death.—The medicinal dose of nux vomica powder is from two to three grains, gradually increased. A long-continued use of it may produce, apparently as a result of accumulation, the effects of chronic poisoning (supra). Large doses are said to have been given, on some occasions, with impunity.

The smallest fatal dose yet recorded, is *three grains* of the alcoholic extract of nux vomica. Two cases of poisoning occurred in London, in 1889, in each of which fifty grains of the powder proved fatal. In one of these, death took place in *an hour;* the druggist who sold the poison said, that he did not think a dose of fifty grains was sufficient to cause death; but a smaller quantity has been known to destroy life. In an old case, reported by Hofmann (1739), and quoted by Christison (901), also by Traill (Outlines, 137), *thirty grains* of the powder, in two doses of fifteen grains each, proved fatal. The poison was given, by mistake, to a girl, æt. 10, laboring under quartan fever, and summis anxietatibus præcedentibus, et ad vomendum conatibus, she soon afterwards died. This is, I believe, the *smallest* fatal dose of the powder recorded. This question became of some importance in the case of Reg. v. Wren (Winchester Spring Ass. 1851). The prisoner was convicted of an attempt to administer this poison in milk: the quantity separated from the milk amounted to forty-seven grains.

Death may occur in from one to twelve hours; but Dr. Christison quotes a case in which a man died in *fifteen minutes* after taking a dose (898). This is probably the shortest period. There are many instances of recovery on record in which early treatment was rosorted to. Sobernheim mentions the case of a young man, who took half an ounce of the powder, and suffered from the usual symptoms: emetics were administered, and he recovered. A second occurred to Dr. Basedow, of Merseburg. A young lady swallowed, by mistake, a tablespoonful (= 360 grains, or three-quarters of an ounce) of the powder; she was almost instantly deprived of the power of walking, and fell down, but did not lose her recollection. There was great difficulty of breathing. Emetics were administered with good effect, and she recovered. A third case is described by Mr. Baynham, of Birmingham. A girl, æt. 20, swallowed half an ounce of the powder. In half an hour the usual tetanic symptoms came on; she was perfectly sensible. In administering remedies, the spasm of the muscles of the jaw was such as to cause her to bite through the cup. The convulsions gradually subsided in about four hours from the first attack; and the next day, although feeble and exhausted, she was able to walk home. (Med. Gaz. iii, 445.) The reporter of this case states, that he has often prescribed a scruple of powdered nux vomica daily, without any injurious effects following! For another case of recovery, in which half an ounce was taken, see Prov. Med. Journal, Jan. 7, 1846, p. 5; and for a case in which this dose proved fatal in seven hours, see Lancet, May 17th, 1856, p. 551. Husemann has collected forty-one cases of poisoning by nux vomica. (Reil's Journal, 1857, 4 H. p. 521.)

Treatment.—The removal of the poison from the stomach by emetics, or the use of the stomach-pump, must be chiefly relied on. Unless these means are employed early, the jaw may become spasmodically fixed, so as to render all efforts at relief unavailing. In general, however, the spasms have intermissions, so that there may be time to apply remedies in the interval. The free use of emetics has been attended with great benefit. It has been asserted, that vomiting does not occur in this form of poisoning; but this is an error (see cases, ante, p. 673).

Analysis.—The seed of nux vomica is hard, brittle, tough, and difficult to pulverize. The powder is of a gray-brown color, like that of liquorice: it is sometimes met with in a coarsely rasped state: it has an intensely bitter taste. It yields to water and alcohol,—strychnia, brucia, igasuric or strychnic acid, and some common vegetable principles. Heated on platina-foil, it burns with a smoky flame. It is colored brown by a solution of iodine. Nitric acid turns it of a dark orange-red color, which is destroyed by protochloride of tin. The aqueous *infusion* or decoction is reddened by nitric acid, and is freely precipitated by tincture of galls. Persulphate of iron gives with it an olive-green tint. These properties are sufficient to distinguish it from various medicinal powders which it resembles. The fine silky fibres which cover the surface of the seed may be identified by the microscope. For this purpose a small quantity moistened with water, should be examined with a power of about 200 diameters. The fibrous nature of the powder will then be apparent. A solution of iodine gives to the fibres a golden-yellow color. There are no starch-granules to be seen in the genuine powder. As it closely adheres to the mucous membrane of the stomach, the powder may be found in the dead body, and separated by washing from the organic matter with which it is mixed. It is quite insoluble in water. Strychnia may be obtained from it by a process described under that alkaloid (post); but owing to the small proportion present, there will be some difficulty in procuring strychnia in a crystalline state, unless from twenty to thirty grains of the powder are obtained.

In the tissues.—Nux vomica bears the same relation to strychnia that opium does to morphia. It is by the strychnia absorbed from the powder as it lies on the mucous membrane of the stomach, that the life of a person is destroyed. Hence the poison, for which we must seek in the tissues, is strychnia (see p. 788). It is a remarkable fact, that in no case of poisoning by nux vomica yet recorded, so far as I have been able to ascertain, has strychnia been found in the blood or tissues of persons poisoned by it. Strychnia has been pronounced to be indestructible, and in all cases to remain in the dead body unchanged and unchangeable; but those who have expressed this dogmatic opinion have not had the candor to test its accuracy by seeking for absorbed strychnia in the cases of poisoning by nux vomica which have come before them.

NUX VOMICA BARK.

This was formerly confounded with the Cusparia or Angostura bark, and has long been known under the name of false angostura. It contains strychnia and brucia, and in its effects on the body, whether in the state of bark, infusion, or decoction, it resembles the seed of nux vomica. About the latter end of the last century, a quantity of this bark was distributed over Europe, mixed with the angostura and cusparia, and numerous fatal accidents occurred before the true nature of the poisonous bark was discovered. Dr. Husemann has given a full account of these cases in Reil's Journal für Toxikologie, 1857, H 4, p. 511. He has collected eleven cases of poisoning by this bark.

The Strychnos Colubrina, or Snakewood of the East Indies is supposed by some to be the wood of the nux vomica, and to produce similar effects. The term Snakewood is applied in the East, to a variety of woods which, when made into cups, impart a bitter taste to water; and the water is then considered to be an antidote to the bites of venomous serpents.

The nux vomica bark is now seldom met with. It is known by the fracture of the bark acquiring a strong red color on being touched with nitric acid. From its infusion or decoction both strychnia and brucia may be obtained.

ST. IGNATIUS'S BEANS.

These are the seeds of the *Strychnos Ignatii*, or the *Ignatia Amara* (Cabalonga). The tree producing them is said to abound in the Philippine Islands. According to Pereira, the fruit has a pyriform shape, and contains twenty of the seeds. They were first made known by the Jesuits, and they were named after their patron. Pelletier and Caventou found that they contained 1·2 per cent. of strychnia: when taken in powder they produce symptoms and effects similar to those caused by nux vomica. They were formerly used as a febrifuge in medicine. A case is related by Husemann, in which a man, æt. 40, took one-half of a bean in brandy, to cure an attack of fever. He suffered from the usual tetanic symptoms, and narrowly escaped with his life. Four other cases are referred to by this writer (Reil's Journal für Tox. 1857, H 4, p.

520.) Mr. Bennett gave half a drachm of the seed procured at Manilla to a dog. In twenty-five minutes the dog was suddenly seized with tetanic convulsions. There was panting respiration, trembling of the muscles, twitchings of the face, and frothy saliva issued from the mouth. There was a remission in five minutes. The animal was conscious. In a quarter of an hour after the first access of the symptoms, there was a general convulsion of the body, under which death took place. No particular appearances were found. The stomach had a pinkish hue, and the powdered seed was found in it. There was congestion of the liver, and the blood was generally liquid. In a similar experiment on a dog with the same quantity of seed, the tetanic symptoms came on quite suddenly in half an hour, and after various attacks the animal died in three quarters of an hour from the time of seizure. (Lancet, Aug. 31, 1850, p. 259; and Braithwaite's Retrospect, 1850, p. 415.) These beans are not easily procured; their effects are identical with those of nux vomica and are due to strychnia. According to some authorities brucia is associated with strychnia.

STRYCHNOS (UPAS) TIEUTE.—(JAVA POISON.)

The plant which yields this variety of Upas poison is the *Strychnos Tieute.* It is a large climbing shrub which grows in Java, and is known under the name of *Tshettik.* It contains strychnia, but no brucia. Its effects are similar to those of nux vomica. It produces tetanus, asphyxia, and death. This poison must not be confounded with the UPAS ANTIAR poison of Java, which, according to Pereira, is derived from a large forest tree (ANTIARIS TOXICARIA), growing to the height of from 60 to 100 feet. The milky juice contains 3·56 per cent. of a poisonous principle, called ANTIARIN.

It was long since pointed out by Sir B. Brodie, that this poison operated by paralyzing the heart. Kölliker and Pelikan have lately investigated the subject, and have come to a similar conclusion; but they have found that its principal action is on the voluntary muscles, and that it is essentially a paralyzing poison. It destroys the excitability of the nervous system instead of exalting it like strychnia. It acts with great rapidity on the heart, stopping its action in five or ten minutes.

The *Tanghinia Venenifera*, or Madagascar poison, is a seed of a brownish-black color, of the size of an almond, presenting a wrinkled surface, with an odor resembling that of violets. The fruit resembles the almond, but is larger. This is used in Madagascar as an ordeal poison. Its effect, according to Kölliker, is to paralyze the heart. The *Ordeal* or *Calabar bean* of Africa, or African poison, from a dose of which Dr. Christison suffered severely, also appears to exert its influence chiefly on the heart. The last-mentioned poisons do not contain strychnia.

STRYCHNOS TOXIFERA—CURARA—(SOUTH AMERICAN POISON.)

According to Schomburgk, this tree grows in Guiana, and furnishes a poisonous juice or extract, which, when mixed with other substances, forms an arrow-poison used by the Indians in killing game, or destroying each other. Various names have been given to this substance, according to the district in which it is prepared; but from the recent investigations of Bernard and Pelikan, it is evident that the poison known under the name of *Ticunas*, *Woorali*, *Oorara*, and *Curara*, does not owe its effects to strychnia, and that the plant or plants which yield it, do not belong to the strychnos tribe. At the same time, among the variety of poisonous extracts used by the Indians, there may be one or more containing strychnia. Martius affirms that the Ticunas extract is derived from the Cocculus Amazonum, and that it contains picrotoxia (p. 662).

The South American poison is now generally known under the name of Curara, from the plant *Curari* from which it is obtained. It contains a poisonous alkaloid, first discovered by Boussingault, in 1828, which is called *Curarina*. Animals have been said to fall instantly dead when shot with an arrow poisoned by Curara, but this has only been in cases where a vital organ like the heart has been wounded, and then death was not due to the poison. According to Mr. Waterton, the poisonous extract is procured chiefly from the bark of a creeper or vine which grows in the forests of Guiana and Central America. The Indians prepare the poison with a great deal of mystery, and mix with it other herbs, red and black ants, and the pounded fangs of a venomous snake. The juice is extracted from the stem of the creeper by infusion and compression; it is then heated with the other ingredients over a slow fire, until it acquires a dark color and an intensely bitter taste. It is afterwards put into a small pot, carefully covered over, and kept in a dry place. It is occasionally warmed over a fire that it may be kept dry.

The extract is so exceedingly miscible with water, that the slightest moisture dissolves it; hence, it speedily diffuses itself when introduced into a wound. The symptoms which it produces in animals are stupor and paralysis. It does not begin to produce any apparent effects until after a lapse of one or two minutes, and there is apparently no pain: convulsions come on in two or three minutes, and the animal dies in four or five minutes. Putrefaction is not accelerated, and the flesh of the game thus killed is used as food without any serious effects resulting. This is probably due to the very small quantity of absorbed poison present; for the Curara, in a sufficient dose, is fatal to all animals. It requires much more of this extract to kill an ox than a smaller animal; thus, the Indian adjusts the size of his arrow and the quantity of poison to the size of the animal. In one experiment, three arrows were introduced beneath the skin of an ox. For four minutes there was no effect: the animal then set itself firmly on its four legs as if to resist falling, and remained quite still for fourteen minutes. It then attempted to walk, staggered, and fell. The eyes became fixed, dim, and apparently insensible to light. Convulsions appeared in the legs; there was emprosthotonos, laborious respiration, and an escape of a frothy liquid from the mouth. The convulsions in the extremities gradually ceased: there was still a perceptible action of the heart at intervals. In twenty-five beyond all doubt, strychnia was present, and that the person must have died of it!

STRYCHNIA AND ITS SALTS.

Remarks.—STRYCHNIA (from *ὁ στρυχνος*, a term applied to plants supposed to have the narcotic properties of nightshade) is an alkaloid extracted from nux vomica. It was discovered in 1818, but it was not until eight or ten years after its discovery that it came into medicinal use. Its properties as a deadly poison have been long known to medical men, but they have only within the last few years been brought prominently before the public, and this alkaloid has now acquired a fatal notoriety. From the time of its discovery up to 1856 it had caused at least *seventeen* deaths, and numerous cases of accidental poisoning from the effects of which the persons had recovered. Husemann, in 1857, had collected thirty-five cases of poisoning by strychnia and its salts; namely, twenty-four with the pure alkaloid, eight with the nitrate, two with the sulphate, and one with the acetate. (Reil's Journal, 4 H. p. 521, 1857.) A large amount of experience has now been collected on this form of poisoning.

SYMPTOMS.—When strychnia is taken in solution it has a hot and intensely bitter *taste*. This, of course, is not necessarily perceived when it is swallowed in the form of a pill. At an interval varying from a few minutes to one hour or longer, and sometimes without any premonitory symptoms, the person is suddenly seized with a feeling of suffocation and great difficulty of breathing. There are twitchings and jerkings of the head and limbs,—a shuddering or trembling of the whole frame. Tetanic convulsions then commence suddenly with great violence, and nearly all the muscles of the body are simultaneously affected. The limbs are stretched out, the hands clenched—the head, after some convulsive jerkings, is bent backwards, the whole body is as stiff as a board,—and assumes, by increase of the convulsions, a bow-like form (opisthotonos), being arched in the back and resting on the head and heels. During the fit the head is firmly bent backwards, and the soles of the feet are incurvated or arched and everted. The abdomen is hard and tense—the chest spasmodically fixed—so that respiration appears to be arrested—the face assumes a dusky or congested appearance, with a drawn and anxious aspect, the eyeballs are prominent and staring, and the lips are livid. The features have been observed to assume the peculiar appearance given by the sardonic grin (risus sardonicus). The patient complains of a choking sensation, with thirst and dryness of the throat. An attempt to drink is often accompanied with a spasmodic closure of the jaws, by which the glass or vessel is broken or bitten. In several cases of poisoning by strychnia there has been from the outset a sense of impending dissolution, and one of the first exclamations made by the patient has been, "I shall die." The intellect is generally clear and unclouded during the intervals of the paroxysms, and the patient appears to have a full sense of his danger. After a succession of fits, and generally shortly before death there may be a loss of consciousness. This was observed in a case which occurred to Dr. Ogston, and in that of Mrs. Dove

Pain is occasionally felt at the pit of the stomach, and during paroxysms there is severe suffering from the violent spasm of the voluntary muscles. The consciousness of the access of the fit is very remarkable. The patient calls out loudly, "It is coming," and screams or shrieks, asking at the same time to be held. He in vain seeks for relief in gasping for air and in requiring to be turned over, moved, or held. Sometimes there is frothing at the mouth, and this froth is bloody from injury to the tongue. With respect to the muscles of the lower jaw—these, which are the first to be affected in tetanus from disease, are generally the last to be affected by the poison. The jaw is not primarily attacked, and is not always fixed during the paroxysm. It is relaxed in the interval, and the patient can frequently speak and swallow. When the jaw has been fixed by spasm, unlike the lock-jaw of disease, this has come on suddenly in full intensity, with tetanic spasms in other parts, and there are intermissions which are not witnessed in the tetanus of disease (see p. 134). The sudden and universal convulsion affecting the voluntary muscles has been sometimes so violent that the patient has been raised up and even jerked off the bed. During the convulsions the pulse is very quick. After an interval of half a minute to one or two minutes, the convulsions subside; there is an intermission—the patient feels exhausted and is sometimes bathed in perspiration. It has been noticed in some of these cases that the pupils were dilated during the paroxysm, while in the intermission they were contracted. Slight causes, such as the attempt to move, or a sudden disturbance, or even touching the person lightly, will frequently bring on a recurrence of the convulsions. In cases likely to prove fatal, they rapidly succeed each other and increase in severity and duration until at length the patient dies exhausted. The tetanic symptoms produced by strychnia, when once clearly established, progress rapidly either to death or recovery. The duration of the case, when the symptoms have set in, is reckoned by minutes, while in the tetanus of disease when fatal, it is reckoned by hours, days, and even weeks. As a general statement of the course of these cases of poisoning,—within *two hours* from the commencement of the symptoms the person either dies or recovers, according to the severity of the paroxysms and the strength of his constitution.

The *time at which the symptoms commence* appears from the recorded cases to be subject to great variation. In poisoning by nux vomica the symptoms are generally more slow in appearing than in poisoning by strychnia. Until they set in suddenly, the patient is capable of walking, talking, and going through his or her usual occupations. On an average in poisoning by strychnia the symptoms appear in from five to twenty minutes. The interval which may elapse between the taking of the poison and the first appearance of symptoms has formed a subject of discussion, and in the case of a notorious criminal it was made a cardinal point of the medical defence. (See ante, p. 114.) Dr. Warner, æt. 39, took by mistake half a grain of sulphate of strychnia; the symptoms began in less than *five minutes*, by constriction of the throat, tightness of the chest, and rigidity of the muscles on attempting to move. He first complained of want of air, and requested the windows to be opened. He died in from fourteen to twenty minutes, his mind remaining clear until the last. (Brit. Amer. Journal, August, 1847.) In that of Mrs. S. Smyth, of Romsey, three grains of strychnia were taken by mistake for salicine. This lady was in violent spasms in from *five* to ten minutes afterwards, and she died in one hour and a quarter. (Pharm. Journal, 1848, vol. ii, p. 298.) A girl, æt. 13, took one grain and a half of strychnia in solution on an empty stomach; the symptoms began by twitchings of the muscles rather more than *an hour* after the poison was taken; and she died in a violent tetanic fit in two hours and a half after she had taken the poison. (Mr. Bennett, in Lancet, Aug. 31, 1850.) This was a case in which, according to theory, the symptoms should have commenced within a few minutes! I have elsewhere referred to the case of Assistant-surgeon Bond (pp. 115, 140). This gentleman took two grains of strychnia at 11·30 P. M., believing at the time that he had taken two aperient pills. He went to bed, and was soon sound asleep. About 1·30 A.M., *two hours* after taking the pills, he started from his sleep, awoke his wife, and said that he should die. Convulsive action of the limbs with difficulty of breathing immediately followed. At 2·20 A.M., he was seen by my informant, and was then suffering from tetanic convulsions. No suspicion existed in the mind of deceased or of any one about him that he had taken poison. He was bled, when a severe convulsion came on suddenly, and he died about three hours after he had taken the pills. This case occurred in December, 1857, and a report was forwarded to me in March, 1858. In a recent case, reported by Dr. Ogilvie, of Alexandria, in which about four grains of strychnia were taken, the symptoms did not appear until *an hour* after the poison was taken. (Med. Times and Gaz. Oct. 30, 1858, p. 443.) Drs. Lawrie and Cowan have reported the case of a medical man who, in June, 1853, took three grains of strychnia, dissolved in rectified spirits and diluted sulphuric acid. He went to bed and slept for about *one hour and a half*, when he awoke in a spasm, uttering loud cries, which alarmed the household. Under treatment, this gentleman recovered. (Glasgow Med. Journal, Part xiv, July, 1856.) The longest interval recorded was in a case which occurred to Dr. Anderson, in 1848, in which *two hours and a half* elapsed before the appearance of symptoms. In this case the man took three and a half grains of strychnia by mistake for muriate of morphia. He recovered. (Ed. Monthly Journal, 1848, p. 566.) Other cases are reported in which the symptoms appeared in from ten minutes to three quarters of an hour. (Guy's Hosp. Reports, Oct. 1856, p. 346; also Reil's Journal für Toxicologie, 1857, 2 H. p. 499.)

Another fact connected with the symptoms worthy of notice is, that there is a great exaltation of sensibility, and sometimes of the senses of sight and hearing; hence a slight touch may induce a tetanic paroxysm. On the other hand, patients suffering from the effects of strychnia have frequently derived great relief from being held, moved, restrained, or rubbed, during the convulsive fit. (See cases, ante, p. 674.) In Dr. Lawrie's case (supra) great relief was given by the forcible extension of the body; and in the cases of Mrs. S. Smyth (supra) and J. P. Cook (post) each desired to be turned over. In fact, while a slight touch, by acting more as an excitant, may induce a spasm, a firm grasp has not produced this effect. (Report on Strychnia, by Dr. Steiner, Philadelphia, limbs, with fixedness of the joints. Among the *internal* appearances are,—congestion of the membranes and substance of the brain, as also of the upper part of the spinal marrow; congestion of the lungs and air-passages; the heart is contracted and empty, but its right cavities, in other instances, have been found distended with liquid blood. The blood is black and liquid throughout the body. The mucous membrane of the stomach has occasionally presented patches of congestion, probably depending on extraneous causes, since, in other instances, the stomach and intestines have been found quite healthy. The bladder is generally empty.

A gentleman who had taken about six grains of strychnia was found dead. The body was examined on the same day. The face was pale, the features were calm and placid, the eyes were closed, the pupils natural. The arms, although still warm, were rigid and bent at right angles, lying across the chest. The whole body was rigid and curved in a state of opisthotonos, resting upon the heels and back of the head. There was some lividity on the right side. The feet were slightly turned inwards. The muscles were of a bright-red color, the lungs were slightly congested, the heart was larger than natural, the right cavities were distended with dark fluid blood, the left cavities contained but a small quantity. The stomach was congested, the mucous membrane presenting some dark-brown patches. It contained about six ounces of food, and in some of the folds strychnia was visible. The liver, gall-bladder, kidneys, spleen, and intestines presented nothing unusual. The bladder was, in this case, nearly full of urine. The brain was slightly congested. (Mr. Porter, in Dublin Hosp. Gaz. Aug. 1, 1858, p. 227; see also paper by Dr. Geoghegan, Dublin Med. Press, June 25, 1856, p. 401.)

In a case in which a person died in six hours from a dose of three grains (p. 668, post), the rigidity of the body seven hours after death was so great as to allow it to be lifted by the heels. It was as stiff as wood, and all the muscles were firmly contracted. Thirty-six hours after death the rigidity had diminished except in the fingers. The lungs were congested; the heart was flabby, the right cavities containing dark-colored blood, partly fluid and partly coagulated. The liquid portion appeared full of air-bubbles. The only appearance observed in the abdomen was congestion of the kidneys. In the head, the membranes of the brain (dura and pia mater) were congested; the substance of the brain was also congested. The ventricles contained much serum; the choroid plexus was congested and of a dusky color. The upper part of the spinal marrow was very red superficially, and the canal appeared to be full of serum. The scalp was loaded with blood. (Guy's Hosp. Reports, Oct. 1857, p. 484.) Mr. Wilkins forwarded the stomach of the deceased to me. The mucous membrane was very rugose, and of a dark brownish-red color. At the greater end there was a red patch, arising from congestion, covering about three-quarters of an inch of the mucous surface. There was a diffused redness of the lining membrane, amounting to deep lividity at the two ends of the stomach. It was softened, and a thick layer of mucus adhered to it. It contained about seven ounces of fluid of a light reddish-brown color. This was removed and rigidity, the hands and feet being incurvated by muscular contraction. The membranes of the brain, especially the inner membrane (the pia mater), were much congested. There was bloody serum beneath this membrane and in the ventricles. The substance of the brain, as well as the membranes and substance of the spinal cord, were congested. The cavities of the heart were nearly empty; the small quantity of blood therein was dark and fluid. The blood was generally fluid, and of the consistency of treacle. The lungs and air-passages were engorged with dark blood, presenting the appearance of pulmonary apoplexy; the mucous membrane of the windpipe was of a dark plum-color, and presented on its surface a dark mucus. The other organs, including the stomach, were healthy. In the stomach there were slight appearances of congestion.

Fatal Dose.—The medicinal dose of strychnia is from the $\frac{1}{20}$th to $\frac{1}{16}$th of a grain two or three times a day, gradually increased to as much as half a grain. The late Dr. Pereira states that he has given as much as one grain and a half at a dose; but subsequent experience satisfied him that so large a dose was dangerous. (Mat. Med. vol. ii, pt. i, p. 654.) Andral found that a dose of $\frac{1}{12}$th of a grain produced, in a man suffering from hemiplegia, intense tetanic rigidity in the paralyzed limb. (Clinique Medicale, by Spillan, 1836, p. 890.) Dr. Christison communicated to me a case in which the $\frac{1}{16}$th part of a grain of strychnia in a pill, killed a child between two and three years of age in four hours. In two cases of adults, in each of which a quarter of a grain had been taken by mistake, the patients only recoverd under early treatment. (Lancet, July 26, 1856, pp. 107–117.) The smallest fatal dose recorded in an adult, was in the case of Dr. Warner. *Half a grain* of the sulphate of strychnia here destroyed life. In another instance, a woman, æt. 22, an in-patient of the Jersey Hospital, had administered to her by mistake four pills, each containing $\frac{1}{8}$th of a grain of strychnia. Soon afterwards, the usual symptoms appeared, and speedily proved fatal. (Dub. Med. Press, Sept. 17, 1852, p. 182.) In this case *one-half grain* of strychnia proved fatal. Three-quarters of a grain have destroyed life in at least three cases. (On Poisoning by Strychnia, pp. 138, 139.) There are at least three instances recorded in which persons have recovered after taking a grain; in two cases, after three grains (p. 683 ante); and, in one instance, a person is said to have recovered from a dose of seven grains. (Medical Gazette, vol. xli, p. 305.) These, however, must be regarded as exceptional cases. A fatal dose of strychnia for an adult may be assigned at from *half a grain to two grains.*

Period at which death takes place.—In fatal cases, death generally takes place within two hours after the taking of the strychnia. One of the most rapidly fatal cases recorded is that of Dr. Warner (p. 682). The symptoms commenced in five minutes, and he was dead in *twenty minutes.* In the case of Cook, the symptoms commenced in an hour and a quarter, and he died in twenty minutes after their commencement. In a case privately communicated to me, ten grains of strychnia, given by mistake for sulphate of quinine, killed a patient in ten minutes. In Dr. Ogston's case, in which three-quarters of a grain were taken, the man appears to have died in less than a quarter of rigidity, the hands and feet being incurvated by muscular contraction. The membranes of the brain, especially the inner membrane (the pia mater), were much congested. There was bloody serum beneath this membrane and in the ventricles. The substance of the brain, as well as the membranes and substance of the spinal cord, were congested. The cavities of the heart were nearly empty; the small quantity of blood therein was dark and fluid. The blood was generally fluid, and of the consistency of treacle. The lungs and air-passages were engorged with dark blood, presenting the appearance of pulmonary apoplexy; the mucous membrane of the windpipe was of a dark plum-color, and presented on its surface a dark mucus. The other organs, including the stomach, were healthy. In the stomach there were slight appearances of congestion.

FATAL DOSE.—The medicinal dose of strychnia is from the $\frac{1}{20}$th to $\frac{1}{16}$th of a grain two or three times a day, gradually increased to as much as half a grain. The late Dr. Pereira states that he has given as much as one grain and a half at a dose; but subsequent experience satisfied him that so large a dose was dangerous. (Mat. Med. vol. ii, pt. i, p. 654.) Andral found that a dose of $\frac{1}{12}$th of a grain produced, in a man suffering from hemiplegia, intense tetanic rigidity in the paralyzed limb. (Clinique Medicale, by Spillan, 1836, p. 890.) Dr. Christison communicated to mė a case in which the $\frac{1}{16}$th part of a grain of strychnia in a pill, killed a child between two and three years of age in four hours. In two cases of adults, in each of which a quarter of a grain had been taken by mistake, the patients only recoverd under early treatment. (Lancet, July 26, 1856, pp. 107–117.) The smallest fatal dose recorded in an adult, was in the case of Dr. Warner. *Half a grain* of the sulphate of strychnia here destroyed life. In another instance, a woman, æt. 22, an in-patient of the Jersey Hospital, had administered to her by mistake four pills, each containing $\frac{1}{8}$th of a grain of strychnia. Soon afterwards, the usual symptoms appeared, and speedily proved fatal. (Dub. Med. Press, Sept. 17, 1852, p. 182.) In this case *one-half grain* of strychnia proved fatal. Three-quarters of a grain have destroyed life in at least three cases. (On Poisoning by Strychnia, pp. 138, 139.) There are at least three instances recorded in which persons have recovered after taking a grain; in two cases, after three grains (p. 683 ante); and, in one instance, a person is said to have recovered from a dose of seven grains. (Medical Gazette, vol. xli, p. 305.) These, however, must be regarded as exceptional cases. A fatal dose of strychnia for an adult may be assigned at from *half a grain to two grains.*

PERIOD AT WHICH DEATH TAKES PLACE.—In fatal cases, death generally takes place within two hours after the taking of the strychnia. One of the most rapidly fatal cases recorded is that of Dr. Warner (p. 682). The symptoms commenced in five minutes, and he was dead in *twenty minutes.* In the case of Cook, the symptoms commenced in an hour and a quarter, and he died in twenty minutes after their commencement. In a case privately communicated to me, ten grains of strychnia, given by mistake for sulphate of quinine, killed a patient in ten minutes. In Dr. Ogston's case, in which three-quarters of a grain were taken, the man appears to have died in less than a quarter of been supposed that emetics would not act in these cases; but this is an error, based on imperfect observation. In Dr. Cowan's case (p. 683), in which three grains of the poison had been swallowed an hour and a half before any treatment could be adopted, free vomiting was induced by the administration of emetics, and this aided in bringing about recovery. (Glasgow Med. Journal, Pt. xiv, July, 1856, p. 4.) Other cases of a similar kind are recorded. In one that occurred to Mr. Hillier, a girl, æt. 18, took two grains of strychnia wrapped in a sweetmeat. In half an hour she felt a drawing of the legs and body; she then took as an emetic some common salt, which did not act for twenty minutes; she then vomited. The stomach-pump was used, and the girl speedily recovered. (Med. Times and Gaz. April 1, 1854, p. 316.) In this instance also much relief was obtained by the use of chloroform vapor to allay the spasms. A case, in which a dose of liquid chloroform is stated to have been beneficial, occurred to Dr. Dresbach (Ohio). A man swallowed three grains of strychnia in three ounces of a solution. He was seen by Dr. Dresbach in twenty minutes, and was then in the following condition: The muscles of the back and legs were rigidly contracted, the features distorted, a sense of burning in the stomach, tightness in the chest, giddiness, dimness of vision, the limbs cold, and the body in a state of copious perspiration. He gave the man two drachms of chloroform, and in less than fifteen minutes after swallowing it the relief is said to have been complete. (Amer. Jour. Med. Sci. April, 1850, p. 546.)

If the poison has been already absorbed and conveyed into the blood, there is no known antidote to its action. Opium has been recommended, but there is no evidence that its employment has been attended with any benefit. Cold affusion has in some cases only had the effect of bringing on a violent paroxysm. A mixture of finely powdered animal charcoal in sugared water has been suggested as an antidote. No injury can follow from its use; but there is no evidence of its having been productive of benefit when it has been administered some time after the poison. This treatment was adopted at St. Bartholomew's Hospital in a case in which a man swallowed four grains of strychnia with an equal quantity of morphia dissolved in spirit. The symptoms came on in half an hour, with a feeling of dread as of some impending danger, and a stiffening of the limbs. He was brought to the hospital an hour after the poison was taken. About three or four ounces of animal charcoal were injected into the stomach by the pump, and the liquid withdrawn was examined by Dr. Stenhouse, but he could find no strychnia therein. The man had the usual symptoms for ten hours; after this he recovered. (Med. Times and Gaz. April 28, 1855, p. 423.) The patient should be kept in perfect quiet and repose so as to prevent as far as possible the recurrence of the paroxysms. The senses are sometimes acutely impressionable. Thus light is intolerable, and the lowest whisper may be heard by the patient. Even the feeling of the pulse or the sudden approach of a person to the bedside has been known to bring on a paroxysm.

Tobacco has been proposed as a remedy, and Dr. O'Reilly, U. S., has published a case in which a man who had swallowed six grains of strychnia recovered after he had taken an infusion of upwards of an residue; the proportion of acid and manganese or bichromate of potash must be adjusted accordingly.

Salts of strychnia in solution—1. If not too diluted, potash and all alkalies throw down a white precipitate not redissolved by a slight excess of the alkali. 2. Carbonate of potash slowly precipitates a diluted solution in fine prisms. 3. Bicarbonate of potash does not precipitate the solution, if acid. 4. Sulphocyanide of potassium produces a crystalline precipitate, appearing as flattened prisms under the microscope. 5. Ammonia gives crystals of strychnia. 6. Chromate of potash produces in very diluted solutions, prismatic crystals of a golden-yellow color, and when a drop of sulphuric acid is added to these under the microscope, the purple and violet colors are immediately brought out. This is a useful test for strychnia, first suggested by Mr. Horsley, of Cheltenham. 7. The chlorides of platina and gold, and the iodide of potassium, with or without iodine, produce precipitates the crystalline characters and polarizing properties of which, as observed by the microscope, are in some respects peculiar. The iodide of potassium gives, even in very diluted solutions, well-marked stellated prisms with fusiform terminations. 8. The solution has an intensely bitter taste, perceptible even when diluted to $\frac{1}{30000}$th part, or according to some, when diluted to $\frac{1}{70000}$th part.

The principal salts of strychnia are the *acetate*, *hydrochlorate*, and *sulphate*. The latter is but little known in this country, and is chiefly used in the United States. All are eminently poisonous. The acids are known by the usual tests. There is at present no restriction on the sale of strychnia or its salts to the public. They may be procured at the rate of threepence a grain, or in larger quantities at sixteen shillings per ounce. Strychnia is largely imported from the Continent, and is exported from England to Australia. It is much employed in the destruction of vermin, and in Australia in the destruction of wild dogs. In 1852, the strychnia annually imported into this country amounted to about fifty ounces. (Pharm. Journal, 1852, p. 538.) A large quantity of strychnia is used in the manufacture of *Battle's vermin powder.* This is a compound of strychnia, starch, and coloring matter (Prussian blue). It is sold over most parts of the kingdom. It has already caused several deaths, and occasioned numerous accidents. (See case, Ed. Med. Journal, Nov. 1858, p. 410.) There is no restriction on its sale. A packet, containing a grain, may be purchased at most drug or oil shops for threepence! As two grains may be purchased (by the ounce) for one penny, and sold as vermin powder for sixpence, the vendors of these dangerous articles realize an enormous profit. Within the last two years, strychnia has been much used for the purposes of suicide.

Objections to the tests.—There are no objections to these tests when we are dealing with the *pure alkaloid*, and when we take care that the more salient properties of crystallization and taste are brought out as well as the production of color. Some enthusiastic chemists have proposed that we should rely on color alone, and they have affirmed that there is no substance but strychnia which will produce, with a mixture of sulphuric acid and bichromate of potash, ferricyanide of potassium, or peroxide of lead, the colors above described. This was stated on of alcohol is added, and the whole is now digested in a water-bath, with frequent stirring. 2. After an hour's digestion, the liquid is strained, filtered, and pressed; the residue is washed with more water and alcohol until all the soluble matter is removed. The liquid is evaporated in a water-bath to one-half, and the residue is then treated with hot alcohol and filtered. 3. The alcoholic solution (containing acetate of strychnia) is evaporated, and the residue is digested with a small quantity of distilled water. This is filtered, and placed in a stoppered tube; it is then rendered alkaline by potash, and shaken with twice its volume of rectified ether. The ethereal liquid is poured off and allowed to evaporate spontaneously, when strychnia, if present, will be left in small circular spots, which appear crystalline under the microscope. The crystalline form, the bitter taste, and the effect produced by sulphuric acid and bichromate of potash, will enable the analyst to determine whether the ethereal residue does or does not contain the alkaloid strychnia.

Among the numerous modifications suggested, the use of chloroform has been recommended in place of ether, and in some cases it may be preferable; but it exerts a powerful solvent action on oil and fat as well as other organic matters, and on the whole, I have not found it to possess any advantage over ether in separating strychnia from organic liquids. It is, however, a better solvent of the pure alkaloid.

In one experiment by the ether process, a quarter of a grain of strychnia in a solution was given to a rabbit; the animal lived twenty minutes, and the amount extracted from the stomach, which contained about half a pound of hay and green food, was probably not more than the $\frac{1}{100}$th of a grain. Drs. Christison and Maclagan, of Edinburgh, adopted Stas's process in a similar experiment on the rabbit, which died from a quarter of a grain of pure strychnia, given in the form of a pill, after the lapse of an hour and three-quarters. Strychnia was found in the stomach by the taste and the bichromate test. (Guy's Hosp. Reports, Oct. 1856, p. 385.) Some processes have been announced as more delicate than that of Stas's; but the so-called discoverers forget that a court of law looks to certainty more than delicacy, and that the alleged power of detecting infinitesimal quantities, although it may temporarily redound to the popularity of the inventor, is of no value at a criminal trial unless the results are such as to leave no reasonable doubt on the mind of independent chemists.

Strychnia does not appear to be materially affected by the decomposition of organic matter. Mr. L. Thompson detected the poison in the remains of a dog far advanced in putrefaction, after four months' burial. I have also detected it in the putrefied stomach of a dog. There is no reason to believe, however, that this alkaloid is as indestructible as arsenic: hence, like other organic poisons, it may disappear after the lapse of years. In the case of Mrs. S. Smyth (ante, p. 683), who died from strychnia in 1848, one-half of the stomach with part of its contents had been buried in a garden. The glass vessel containing it was sent to me by Mr. Taylor, of Romsey, in the spring of 1858. On examining the remains by Stas's process, there was no trace of strychnia. No communicated to me by persons on whose judgment reliance may be placed, are these:—

1. That strychnia may be found in the stomach as in other cases of poisoning, when it has not been entirely absorbed, and the stomach and contents have been properly preserved for analysis. 2. That a putrefied condition of the body does not interfere with its detection and separation when present in the remains. 3. That in some cases, when given in small doses, and in other cases, even in large doses, although it may be detected in the stomach (if carefully preserved), it cannot be detected in the absorbed state in the blood and tissues. 4. That there are no facts derived from experiments on animals, or from observations on the human body, to justify the statement, that in *all* cases of poisoning by strychnia the poison must by proper chemical processes be certainly detected. 5. That in strychnia poisoning, as in morphia and other forms of poisoning, a person may live a sufficient time for the poison to be entirely removed from the stomach, and in this case he may die without a trace of strychnia being found in the blood, tissues, or any part of the body.

As an instrument of murder, if we except the case of Miss Abercromby (1830, p. 135), strychnia had not been known in this country until the crimes of William Palmer, in 1855–6, drew general attention to this poison. The alkaloid had been used for destroying vermin, but it was scarcely known to the public as a deadly poison. Since that date there have been more deaths from strychnia, as the result of suicide or accident, than had been previously recorded. In Canada and the United States it has been employed for criminal purposes. In 1851, a woman named Azenath Smith, was tried in Canada for the murder of John Freeman, by poisoning him with strychnia. (Brit. American Journal, July, 1851; and Med. Gaz. vol. xlviii, p. 517.) The prisoner had cohabited for a short time with the deceased, and the motive assigned for the crime was a desire to possess his property. On the Friday before his death, deceased was suddenly seized with stiffness of the limbs; his legs were stretched out with violent cramps; he could not bear any one to touch him; he perspired greatly. He retained his intellect throughout: he recovered in about an hour, and said that he never had had such cramps before. On the evening of the next day, about ten o'clock, he was seized with similar fits, which continued at intervals through the night; he shook all over his body, his limbs became stiff, and he perspired greatly. Deceased ascribed his attack to some pills which he had taken. He was better on the Monday, but after taking, as he supposed, another dose of the pills, he was seized with jerking of the limbs, twisting of the ankles, and other symptoms of a tetanic character, his head being drawn right back, and his legs straightened out stiff. He said he should die. A medical man was sent for, but he did not arrive until after his death: hence this record of the symptoms immediately before death is known only from the evidence of non-professional bystanders. It was stated that deceased asked for something to take the taste of the pills out of his mouth,—that after the last dose the cramps came on very suddenly, and that he soon died. From the evidence at the trial, it appeared that after the first slight attack, he was seen by a medical man, to whom he described the spasms like "shocks of electricity." The prisoner, who was present, said, in reply to an inquiry, that he had taken two "balm of life pills." On his second visit, the medical witness found him dead, his body was bent back (opisthotonos), and in a state of general spasm. He suspected death from strychnia. The body was buried, but subsequently exhumed and examined. The legs were in a state of rigid spasm, the soles of the feet much arched, the toes drawn forcibly towards the back of each foot; the heart was healthy, but empty in all its cavities; the other organs of the chest and abdomen presented nothing unusual. An alcoholic extract of the contents of the stomach gave, with sulphuric acid and peroxide of lead, a violet color indicative of the presence of strychnia. The prisoner was supposed to be connected with the act by evidence that either she or a person like her, had purchased five grains of strychnia in a neighboring town on the morning of deceased's death. She had asked for and procured preserves to give to the deceased, with his pills. She had predicted he would not recover. She was well acquainted with the properties of strychnia, and had conversed about the destruction of vermin with it. This, indeed, was the reason assigned for the purchase. She ascribed the deceased's death to apoplexy, had the body buried quickly, sold off some of deceased's property, and left the house. The jury were not satisfied of the guilt of the prisoner, and she was acquitted. That deceased died from strychnia was abundantly proved; but the jury may have supposed that he took the poison by some accident, or with the intention of destroying himself.

At Chicago Circuit Court, December, 1854, and January, 1855, G. W. Greene, a banker, was charged with the murder of his wife by strychnia. He had been married for many years, and the general evidence showed that he had latterly maltreated the deceased. It seems that she died suddenly while in apparently good health, and no person witnessed her death. The prisoner assigned the cause to cholera, and made some contradictory statements. An inspection of the body by Drs. Myers and Freer revealed no cause of sudden death: there was no disease. There was lividity about the throat and eyelids, the tongue was protruded, and the limbs were in a rigid state. The stomach, intestines, and brain were healthy. The lungs and heart were also healthy; but the heart was empty, as well as the great vessels near it. Dr. Blaney, a chemist, deposed to finding strychnia in the contents of the stomach of deceased. He employed Stas's process, and estimated that the whole quantity which he found amounted to the twentieth part of a grain. He used the color tests, and, having produced some of the crystalline matter in court, he applied the tests for the satisfaction of the jury: one gave the result indifferently, and the other not at all. The crystals had a bitter taste. It was proved that, in the drawer of a bureau of which the prisoner had the key, strychnia was found: and it is stated that in the same bureau quinine was kept, which deceased was in the habit of taking for ague, from which she suffered. It was also alleged, that deceased herself had used strychnia for poisoning animals. The defence, therefore, turned upon the possibility of deceased having taken the poison (if taken at all) by some mistake for quinine. Nevertheless, the prisoner was convicted. A new trial was moved for and granted.

There may have been other facts of a moral kind which led to the verdict in this case; but, viewing them as they have been here reported to me, there was a failure of the medical evidence to show that death was caused by strychnia. Nothing was known concerning symptoms; the appearances were of a negative kind, and the chemical analysis, admitting it to its fullest extent, merely proved the existence of a medicinal dose of strychnia. In fact, but for this chemical evidence, there would have been no case against the prisoner; and this evidence, in a scientific point of view, was not satisfactory.

The case of Reg. v. William Palmer (Cent. Crim. Court, 14th to 27th May, 1856), has been already adverted to in various parts of this volume. This prisoner was indicted for the murder of his friend, John Parsons Cook, by strychnia, and, after a lengthened trial, was found guilty of the crime. Whether we view the social position of the accused, the enormity of the crime, the stealthy and artful manner in which it was perpetrated, and the unscrupulous efforts made by some professional men to save this notorious criminal from the scaffold, this case surpasses all others in ancient and modern records. The deceased, æt. 28, enjoyed good health, had never been subject to fits and convulsions, and was fond of outdoor sports. On the evening of the 14th November, 1855, while in the society of Palmer, he was first seized with a violent vomiting after drinking some brandy and water. From this illness he recovered, but he had repeated attacks of vomiting after taking various articles of food, until his death, on the night of November 20th. During this time, Palmer was daily with him, and sent him broth, which caused vomiting. Some of this broth had been privately taken by a servant before delivering it to deceased, and it caused her to vomit. The vomiting continued more or less daily after deceased had taken various articles of food, except on November 19th, on which day the prisoner was absent. On the 17th November an aged practitioner (Bamford) was called in by Palmer to attend Cook. On the 18th, Palmer wrote to a medical friend of deceased's (Jones) to come and see him, stating what, as a medical man, he must have known to be untrue, that he was laboring under a bilious attack combined with diarrhœa. Bamford prescribed two pills containing calomel, rhubarb, and half a grain of the acetate of morphia, to be taken at night. They were taken by deceased on the nights of the 17th and 18th, with benefit. On the night of the 19th, at about 10·30, Palmer gave to deceased two pills, and left him. At a quarter before 12, the deceased was heard to scream, and he was then seen by a servant sitting up in bed and beating the bed. He said to this witness, that the pills which had been given to him by Palmer had caused his illness. She thus described his symptoms: his head was in motion, jerking backwards; his arms were straightened out, and his legs were quite stiff; the eyes were staring; the head was drawn back; the mouth closed. He could speak, and he said he should die. The prisoner, who had been sent for at the request of deceased, gave him a wine-glassful of a brown liquid, after which he vomited, and asked to have his hands rubbed. These were stiff, cold, and damp. On November 20th Palmer was with him, and gave him coffee, which he vomited. Jones arrived, and saw him in consultation with Bamford and Palmer in the evening; he was then going on satisfactorily. He refused to take more pills, but it was agreed that he should have the morphia pills that night. The prisoner, Palmer, did not inform either Jones or Bamford, that deceased had been attacked with tetanic spasms on the previous night after having taken pills, and that he, the prisoner, had attended him, and sat with him for several hours. On the night of the 20th, Palmer called on Bamford for the pills: on the previous night, they had been sent by a messenger. Bamford made them up in Palmer's presence, and at *his* request wrote a direction on the box "night-pills," and delivered them to Palmer, who took them with him. Bamford did not again see the deceased alive.

About a quarter past 11 on this night (and about an hour after the pills had been delivered to him by Bamford) the prisoner Palmer gave two pills to deceased in the presence of Jones, calling Jones's attention to Bamford's handwriting on the box. Palmer then left the house. Deceased, fearing an attack like that of the preceding night, requested Jones to have a bed made up, so that he might sleep in the room with him. But for this circumstance, on which the prisoner had not calculated, it is probable that deceased would have been found dead in his bed, and no accurate history of the symptoms preceding death would have been made known. Three-quarters of an hour after taking the pills deceased appeared comfortable, but in ten minutes more (fifty-five minutes after taking them) Jones was suddenly roused by deceased, who was sitting up in bed, said he was going to be ill, asked his friend to rub his neck, and to send for Palmer. After swallowing two other pills (said to contain ammonia), which Palmer had brought with him, deceased fell back on the bed in convulsions. He said he should be suffocated. They tried to raise him, but he was so stiffened out with spasms that it was impossible. He then said, "Turn me over." He was turned on his side, and he died in a few minutes. Jones described the symptoms as those of tetanus; every muscle of the body was stiffened. When his neck was rubbed, the muscles of the head and neck were found to be affected with violent spasms; his head was thrown back; his hands were clenched; and his arms were in a state of rigidity. His jaw was fixed and closed. His body was stretched out, and rested on the head and heels (opisthotonos). The symptoms, therefore, in this second and fatal attack, came on in about an hour after deceased had taken the suspected pills, and he died in from sixteen to twenty minutes after their commencement.

The body of deceased was inspected on the 26th Nov., six days after death; it was then in a state of rigid spasm, and this state of spasm continued in the limbs for more than two months after death, *i. e.* when the body was exhumed for a second inspection. The viscera were stated to be universally in a sound and healthy condition. The membranes of the brain were a little congested, the heart was empty, and the blood generally dark and fluid. The mucous membrane of the stomach, as well as that of the intestines, was partially congested. There was no appearance of any disease to account for death.

The stomach and intestines were delivered in a jar to Dr. Rees and myself for analysis, without any other information than that it was suspected the deceased might have died from poison. We could procure no history of the symptoms preceding death. As there has been much misrepresentation regarding the condition of the articles for analysis, I here give parenthetically the facts.

[On opening the jar we found the stomach cut open from end to end, turned inside out, with its mucous membrane lying in contact with the intestines. A short written memorandum, in the form of a private note, was handed to us with the jar; there was, however, no authentic report of the inspection signed by those who were present, and no statement to explain the extraordinary and improper condition in which, in a suspected case of poisoning, the stomach had been sent to us. There was no intimation that any contents had been found in the stomach, or, if found, of what had become of them; and it was not until the trial of the prisoner, six months afterwards, that Dr. Rees and I could arrive at a solution of the mystery.

The prisoner, Palmer, threw every difficulty in the way of an inspection. Without waiting for the authority of the relatives of deceased, he ordered a coffin, and it was only by the timely arrival of the stepfather, Stephens, and the suspicion which arose in his mind from the general conduct of the prisoner, that the body was not buried. It subsequently transpired that the criminal, Palmer, had attended the inspection, which was performed by two young men, to one of whom (Newton) he had just before given two wine-glassfuls of brandy. Newton, it is stated, had never before made the inspection of a body—he was on intimate terms with the prisoner; he had secretly supplied the prisoner with three grains of strychnia on the night of the 19th Nov.—the first night on which deceased was attacked with symptoms of poisoning—a fact which he kept concealed for six months, *i. e.* until the trial. He was cognizant of the purchase by the prisoner of six grains of strychnia on the 20th Nov., the day on which Cook died suddenly. On the evening of the 25th Nov. he discussed with the prisoner the fatal dose of strychnia, and whether this poison could or could not be found in the body after death; and on the morning of the 26th, under the circumstances described, he assisted at the inspection of the body of Cook. His co-inspector, Devonshire, published, after the trial, the following account of Newton's proceedings: "He (Newton) punctured the stomach, and about a teaspoonful of the contents were lost. Afterwards, when Dr. Harland and I were examining the lining membrane, Mr. Newton *suddenly turned the stomach inside out;* and an additional half teaspoonful was thus lost, the remainder falling into the jar." (Letter to the Times, May 29, 1856.) It is an anomaly in medico-legal practice in this country to find a professional murderer attending an inspection of the body of his victim, and the inspection conducted by a friend who had secretly supplied him with the poison, and had already discussed the probability of finding it in a dead body.

Under these circumstances it is not straining an inference too far to assert that, if such a practice were common in cases of poisoning by the vegetable alkaloids, we might look in vain for the material proof of the presence of poison. It transpired too that, after the mangled stomach and intestines had been placed in the jar, the criminal Palmer was able to elude the vigilance of the inspectors by running off with the jar and cutting through the bladder with which it was secured. It was stated by

one of the witnesses at the trial that the stomach was tied up where it was punctured by Newton; but if so, Palmer or his friend Newton, the co-inspector, had certainly removed the ligature and spilled the contents, since the stomach, as delivered to us, was cut in various directions, the mucous membrane was smooth and free from any contents, and there was no appearance of any ligature. Such a state of things displayed either deplorable ignorance in managing an inspection, or the greatest criminality in an endeavor to defeat chemical evidence. Dr. Rees and I were not made aware of these facts until at and after the trial, or they would have been made a subject of comment and exposure at the inquest held six months previously. Indeed, it was studiously concealed from us at the inquest that Palmer and his friend Newton were present at the inspection,—that Newton had destroyed the stomach and spilled the contents,—and that Palmer had cut the coverings of the jar after they had been secured! A second jar was sent to Dr. Rees and myself. This contained the liver, spleen, kidneys, with a part of the lungs, the whole mixed together, and in the midst of these viscera, turned upside down, was a corked bottle containing some blood! No memorandum accompanied this jar, and we could procure no information respecting the contents further than that they were taken from the body of Cook. The chemical witnesses retained for the defence of the criminal at the subsequent trial dealt with this inspection as if it were nothing out of the common way, and stated on oath that the mangling of the stomach and spilling of its contents could have created no difficulty with respect to the results of the analysis. This question may be safely left for decision to those members of the profession who do not trade in evidence, and who have had some experience of the chemical analysis of the vegetable alkaloids.]

Dr. Rees and I made an examination of the coats of the stomach and of the coats and contents of the bowels. The only poison found in these and other organs was antimony in traces, and this discovery at once explained the cause of the vomiting from which deceased had suffered during his illness. No antimony had been prescribed for him by his medical attendant.

As there were no contents of the stomach discoverable in the jar, we examined the coats for various poisons—among others for strychnia—by the process known as that of Merck, and described by Fresenius. (Qualitative Chemical Analysis, by Bullock, p. 323.) We preferred this process for its simplicity, and although not so delicate as that of Stas, yet it has this advantage: it separates strychnia (if present) in a form to leave no doubt upon the mind. It was this process which was used by Dr. Ogston in his case (ante, p. 686), and by Mr. Morley subsequently, in the case of Dove (p. 703). It has been ignorantly attacked by those who for a time had a personal interest in attacking it, and who would have equally attacked any process whatever that Dr. Rees and I had adopted. We found no bitter taste in the alcoholic extract, and no satisfactory indication of the presence of strychnia. The charcoal process of Graham equally failed to show the presence of the poison. There was an effect produced by one color test, which would probably have satisfied some ardent chemist of the undoubted presence of strychnia. We, however, declined to risk the possible conviction of a man for murder upon so slender a piece of evidence as this. When we were at length furnished with an account of the symptoms under which deceased had died, we did not hesitate to refer death to strychnia, in spite of these equivocal chemical results: and this opinion was subsequently confirmed at the trial by the evidence of some of the most eminent pathologists and physiologists of the day, including Brodie, Todd, Christison, Curling, Solly, and others.

It may be sufficient to state that the moral evidence against the prisoner was of the strongest possible kind. He had been associated with the deceased in various money transactions connected with racing, to such an extent that the death of the deceased had become, at that time, a necessity to him, in order, as he thought, to extricate himself from his difficulties.

The medical evidence for the prosecution was to the effect that viewing the symptoms as a whole in the two attacks, they were unlike any known form of disease, and admitted of no reasonable explanation, except that of death from strychnia. The grounds of distinction between them and the symptoms of tetanus from disease, have been elsewhere fully detailed (ante, p. 137). It was proved that the prisoner had secretly possessed himself of three grains of strychnia on the night of the 19th November, shortly before he gave to deceased the pills which led to the first attack, and that on the 20th, the day of deceased's death, he had procured six grains of strychnia at a druggist's shop. No reasonable motive could be suggested for his procuring nine grains of this poison from two different sources within twenty-four hours, or any explanation given of what had become of it. The prisoner had, therefore, the motive, the means, and the opportunity, of perpetrating the crime, while death by suicide or accident was wholly inconsistent with the facts.

The defence turned mainly on the non-discovery of strychnia in the body. The criminal tampering with the stomach, was conveniently ignored: and it was assumed that no difficulties had been placed in the way of the analysis. With this assumption it was alleged that no person can die of poison unless the poison is found in the dead body, and that strychnia being susceptible of detection up to the minutest fractional part of a grain, its absence, under a proper chemical research, was a proof that deceased could not have died from its effects. To support this view, the counsel for the defence called Mr. Herapath and Dr. Letheby, who asserted their power to detect strychnia up to the one fifty-thousandth part of a grain or less! But while these witnesses, by their chemical evidence, were thus leading the jury to believe that the deceased had *not* died from strychnia, because this substance was not found in the *coats* of his stomach and intestines, they each had a mental reservation to the effect, that the non-detection was not really owing to the absence of the poison, but to the alleged imperfect process pursued by the crown witnesses for its separation! Mr. Herapath had, indeed, expressed this opinion openly on various occasions before the trial, and Dr. Letheby subsequently published his views to the same effect. If these witnesses had only candidly stated this at the trial, it would have saved the court and jury much time, and science much scandal; for they appeared to differ from the crown witnesses on the main fact, namely, the *cause of death*, when the difference in reality was as to the relative value of their own and other processes for the detection of strychnia,—a point which a jury could not decide, and which was quite unimportant to the issue.

The state in which the stomach was delivered for analysis would to most scientific persons have alone sufficed to account for the failure of the chemical evidence (p. 699); but it was impossible to look for any charitable consideration from men who were bent on making a trial for murder a scene of personal contention and rivalry. Had even the stomach and its contents been delivered to us in an entire state, and the poison not found, the medical dogma on which the defence was based is utterly false. In nearly every chapter on every poison in this volume, the reader will find that chemistry has in some cases completely failed to reveal the presence of poison, while in others it has misled an "expert" to swear to the presence of poison in a definite quantity in a dead body when the whole was a fiction of the imagination (pp. 455, 559).

The evidence for the defence had this bearing. If strychnia had been found in the stomach the death of the deceased would have been at once explained; hence the symptoms taken as a whole were certainly not inconsistent with poisoning. This in fact was admitted by more than one witness for the defence. It was suggested that the symptoms were too long a time in appearing, to have been dependent on the pills—a suggestion utterly at variance with facts (ante, p. 683),—also that the deceased if suffering from the effects of strychnia poisoning could not have borne to be rubbed, and that the cavities of the heart after death would not have been found empty. These statements are all contrary to fact (pp. 683, 704). In short, no natural form of disease could explain the symptoms or death of Cook; and when closely examined, there was not a single incident in the case which was not reconcilable with death from strychnia. The possession of the poison, and the moral circumstances were conclusive of the guilt of the prisoner, while the only point that might have created doubt—the non-discovery of poison—admits of explanation either on the theory of the prosecution or on that of the defence.

On the theory of the chemical witnesses for the defence, a sufficiently delicate chemical process had not been pursued, while on the theory of those for the prosecution, the criminal Palmer and his friend Newton had either ignorantly or designedly destroyed the stomach and its contents, so as to render the detection of a small residuary quantity of this poison impossible. But the view of the witnesses for the defence, when taken with the medical evidence, fully justified the verdict of the jury. A criminal is not to be acquitted upon the assumption that a more delicate chemical process might have been adopted by the crown witnesses for the detection of poison in a dead body; for there is not a criminal case in which an unscrupulous solicitor might not procure this kind of evidence of opinion in favor of the most accomplished professional poisoner. There are various methods of arriving at the same chemical result, and every analyst thinks his own process the best. In fact, the chemical witnesses for the defence differed among themselves as to the best process for extracting strychnia; and they only agreed, *pro hâc vice*, in condemning that which was adopted by the crown witnesses! The jury meanwhile decided the case on the common-sense principle, that evidence based on the 50,000th of a grain of something said to be strychnia by one or two chemists, could not materially add to the value of the evidence from symptoms. Either the symptoms were safe for their guidance without this refined arithmetical addition, or they were not. If they were not safe without it, they could hardly have acquired safety with it, especially when it is considered that the most confident of chemical witnesses are liable to be deceived by the results of their tests (p. 559).

That the prisoner was guilty of the foul crime of murdering his friend, no one who views the whole case apart from prejudice can entertain a reasonable doubt. A distinguished German writer who has commented on this trial, expresses his astonishment that any professional men could be found in England who could stand forward and publicly state on oath that the symptoms under which Cook died might be explained by any form of nervous disease, epilepsy, or angina pectoris. (Dr. Husemann in Reil's Journal, 1857, 4th Heft, p. 564.) It argues but little for the knowledge or moral feelings of medical witnesses, and must shake the confidence of the public, as it has already done to a great extent in the trustworthiness of medical opinions. Such must be the result when scientific witnesses accept briefs for a defence; when they go into a witness-box, believing one thing, and endeavor to lead a jury by their testimony to believe another,—when they make themselves advocates and deal in scientific subtleties, instead of keeping to the plain truth. Such men should be marked by the public, and their efforts at endeavoring to confer impunity on the foulest crimes, and to procure the acquittal of the most atrocious criminals should be duly noted. The chemical defenders of the culprit Tawell on the "apple-pip" theory (ante, p. 602), were in the foremost rank to defend the culprit Palmer! Fortunately for society, their efforts did not prove successful in either case. In the meantime this pernicious system is a heavy blow and a great discouragement to the detection and exposure of murder by secret poisoning. No man in this country can henceforth venture to denounce a grave crime of this kind committed by a person of wealth or of social position, without being prepared to incur the most calumnious attacks, and to have his opinions and motives grossly misrepresented. If, after due consideration, he boldly expresses his opinion at an inquest and persists in it, he is said to be prejudiced; if he hesitates or expresses himself timidly, he is not to be trusted! There is but little protection afforded to a witness by a court of law; the accused person is there the sole object of sympathy and consideration, and a learned counsel is only mildly rebuked who, against the whole bearing of the scientific evidence, asserts that the prisoner is innocent, and asks the jury to adopt his venal assertion in preference to the unbiassed opinions of medical men.

The case of Reg v. Dove (York Summer Assizes, 1856), presents many features of interest. The prisoner was charged with the murder of his wife, by administering to her strychnia. About six days before her death, after having breakfasted with the prisoner, the deceased was suddenly seized with loss of power in the legs, general stiffness, twitchings and cramps of the muscles. These symptoms occurred with greater or less severity during the week, and each attack followed soon after the prisoner had administered medicine to her. She had five or more similar attacks in the six days, but from these she recovered. They were set down to hysteria by the medical attendant, and were treated accordingly. On the evening of her death the prisoner, while partially intoxicated, gave to her some medicine as usual (in a liquid form). She complained of its being hot and very bitter. In less than half an hour she had another attack, and after a succession of fits she died in two hours under all the usual symptoms of tetanus from strychnia. It seems that in every attack, excepting the last, she had asked to be rubbed; the rubbing seemed to relieve her, and on this occasion when she felt the spasm coming on she asked one of her attendants to take hold of her hand. The appearances in the body (see p. 686) corroborated the medical inference, that death had taken place from strychnia, a view supported by Dr. Christison, Mr. Hey, Mr. Morley, and others. Mr. Morley carefully removed the stomach and contents, and in the latter he found enough strychnia not only to give the chemical results with all the tests, but to poison several animals. Traces were also found in the contents of the intestines. For some unexplained reason, the *tissues* were not examined for absorbed strychnia. Considering that at this time accurate scientific information was required on this point, the omission was a serious one, and equally damaging to the cause of justice as well as the interests of science. If there be any truth in the doctrine of the deposition and retention of strychnia in the organs, this woman's body must have been saturated with the poison.

In the defence it was not denied that death had taken place from strychnia. It was proved and admitted that the accused had procured at different times, recently before the deceased's death, ten grains and five grains of strychnia. The former quantity had been used by him, at least in part, to poison cats and mice; the latter quantity was probably that which, in divided doses, had led to the death of his wife. A defence of insanity was set up, but this utterly failed, as a criminal motive, means, and opportunity were apparent; and with a full knowledge of the effects of the poison, there was evidence of conversation on the part of the prisoner regarding the power of detecting the poison in the body. The only strong point of the defence turned upon the non-analysis of the tissues. The slighter attacks of spasms up to the Saturday were assigned to hysteria; they had been treated as such by the medical attendants; if due to strychnia administered in divided doses over a week, the poison would have been found in the tissues, and should have been sought for there. The fatal illness and death, as well as the appearances in the body, and the discovery of strychnia in the contents of the stomach and bowels, were, it was contended, reconcilable with the hypothesis of *one* accidental administration of the poison in the medicine or food on Saturday night. The supposition of accident was however inconsistent with the conduct of the prisoner, and he was properly convicted and executed. Nevertheless it is a matter to be regretted that the alleged frequency of administration was not supported by the detection of strychnia in the blood and tissues, especially as one of the analysts (Mr. Nunneley) had volunteered his opinion at the trial of Palmer three months previously that it might be there detected! But considering the kind of defence set up in Palmer's case, and supported by Mr. Nunneley, the omission was judicious. The result might have at once falsified some of the loose assertions made at this trial.

BRUCIA.

This is an alkaloid generally associated with strychnia. The seeds of the nux vomica yield chiefly strychnia, while the bark of the tree is said to contain brucia in large proportion. It derives its name from Bruce, the Abyssinian traveller, as it was extracted from the bark of a tree supposed to have been discovered by Bruce in Abyssinia, whereas the bark turned out to be that of the nux vomica from the East Indies. Brucia is also found in St. Ignatius's beans.

Symptoms and Effects.—This alkaloid and its salts produce in man and animals symptoms similar to those caused by strychnia. It is not so powerful a poison, and requires to be given in much larger doses. Magendie regarded it as having one-twelfth of the strength of strychnia, while Andral assigns to it one-sixth of the power. As a medicine, it may be given in doses of half a grain, a quantity which would prove fatal if strychnia were employed. Poisoning by brucia is rare. Casper refers to three cases of death from rat poison, containing arsenic and brucia. No trace of brucia was found in the stomach. (Ger. Med. 1857, p. 444.)

Analysis.—Brucia is much more soluble in water and alcohol than strychnia. It may be separated from strychnia by alcohol. Its hot aqueous solution has a strong alkaline reaction. It is easily dissolved by dilute acids. 1. By dissolving it in hydrochloric acid, and adding ammonia, it may be obtained in groups of stellated crystals. 2. Sulphocyanide of potassium separates it from its solutions in crystalline tufts. 3. Nitric acid gives to brucia and its salts, either solid or in solution, a deep blood-red color. 4. If the liquid thus reddened be gently warmed, and chloride of tin is gradually added to it when cold, it assumes a rich purple-red color. An excess of the chloride of tin or nitric acid will destroy this color. 5. Strong sulphuric acid colors brucia of a rich rose-pink color; on adding to this mixture bichromate of potash, oxide of manganese, ferricyanide of potassium, or peroxide of lead, the blue, violet, and purple colors, observed in experimenting on strychnia, are not produced. The mixture slowly acquires an olive or greenish-brown color. 6. Chromate of potash does not act upon a solution of brucia as upon strychnia. 7. The solutions have a bitter taste; they are precipitated by potash and other alkalies.

CEREBRO-SPINAL POISONS.

CHAPTER XLIV.

GENERAL REMARKS—COMMON HEMLOCK—SYMPTOMS AND APPEARANCES—CONIA—ITS PROPERTIES—WATER HEMLOCK—ŒNANTHE CROCATA—ÆTHUSA CYNAPIUM—MONKSHOOD—SYMPTOMS AND APPEARANCES—ACONITINA—BELLADONNA—ATROPIA—LOBELIA—FOXGLOVE—DIGITALIA—DATURA—DATURIA—LABURNUM—CYTISINE—YEW—PRIVET.

Remarks.—THE poisons belonging to this class are considered to affect both the brain and spinal marrow directly, and the heart and other organs indirectly. They are derived chiefly from the vegetable kingdom, and owe their noxious properties to the presence of an *alkaloid*, which admits of separation. The effects of the alkaloid are not only more intense than those of the vegetable from which it is extracted, but sometimes different in kind. Thus the vegetable may produce irritation, and inflammation of the stomach and bowels, before the cerebro-spinal symptoms are manifested, while the alkaloid may operate immediately on the brain and spinal marrow. Among the alkaloids, conia and aconitina are the most powerful. In some cases they appear to act as spinal poisons, for the intellect has remained unaffected. Conia operates in two opposite modes on the spinal marrow,—it produces a state analogous to tetanus, or more commonly it causes a complete paralysis of the muscular system without materially affecting sensibility. It also acts locally as an irritant. The term narcotico-irritant, however, is not appropriate to this class of poisons: for many of them operate without causing stupor or insensibility; and so far as the alkaloids are concerned, there is no marked degree of irritation on the stomach or bowels.

The symptoms caused by the leaves or roots of the vegetable poisons, are generally manifested within an hour: those caused by the vegetable alkaloid,—within a few seconds or minutes. As a summary, the symptoms from this class of poisons may be stated to be giddiness, delirium, coma, paralysis of motion or sensation, or both, with convulsions of a clonic or tetanic kind. The pupils of the eyes are commonly dilated. Occasionally there is vomiting, and severe pain in the stomach and bowels. The poisons in the state of leaves or root have in general a hot, bitter, or other well-marked taste, so that they cannot be criminally administered without exciting suspicion. Murder by monkshood has, however, been perpetrated by the substitution of the leaves of this plant for other vegetables at a meal, and in one instance in which I was consulted, a child was destroyed by sipping a decoction of hemlock. Fatal accidents have occurred by reason of persons eating the roots of the œnanthe crocata, the taste of which they found palatable,—the plant having been mistaken for wild celery or wild parsnip.

As there is a great variety in the effects produced by this class of poisons, so the appearances in the bodies of those who have been killed by them have been subject to variation. In some instances the stomach and intestines are congested or inflamed: in others not. When the person has died under symptoms of narcotism, traces of cerebral congestion are occasionally found, and when the symptoms have been those of asphyxia from paralysis of the muscles of the chest, there has been congestion of the heart and lungs. Microscopically the blood is not altered, but it is generally dark and liquid.

Analysis.—Most of the cerebro-spinal or narcotico-irritant poisons owe their deleterious effects to the presence of an alkaloidal principle similar to morphia, and susceptible of insulation by complex chemical processes. There is, however, considerable difficulty in extracting these alkaloids from the respective vegetables; and when extracted, the chemical differences among them, in respect to the action of tests, are very slight. Indeed, better evidence of the poisonous nature of a suspected liquid has been hitherto derived from the exhibition of a portion of it to animals, than from the application of chemical tests. In a medico-legal point of view, there are, with few exceptions, no chemical tests for these vegetable alkaloidal poisons, in organic liquids or solids, upon which reliance can be placed. The pretenders to minute accuracy of analysis have applied their tests to the isolated alkaloid, and have led the public to believe that it was just as easily tested in the absorbed state in the blood and soft organs of the dead body. Experience, however, shows the contrary. When the vegetable has been used, either in the shape of seeds, leaves, berries, or root, then valuable evidence may be sometimes procured by searching with or without the aid of a good microscope for the botanical characters of the plant; these parts of the plant, from their indigestible nature, may be found in the vomited matters or evacuations during life, or in the stomach and bowels after death. The broken leaves may be separated by washing, as they are quite insoluble in water; they may be therefore easily collected, dried on mica and examined by the microscope, which, under the hands of a skilful botanist, may thus reveal the nature of the poison. This source of evidence will, however, often fail, owing to the poison having been taken in the form of extract, infusion or decoction.

Treatment.—The treatment of a case of cerebro-spinal poisoning consists in promoting early vomiting by emetics, or in drawing off the contents of the stomach when this is possible by the stomach-pump. If there should be reason to suppose, from the seat of pain, that the poison has descended into the bowels, then laxative injections or castor oil may be used. Recoveries have taken place when the poison has been thus removed, even although symptoms had set in. Cold affusion, or stimulants, may occasionally be required; the patient, if inclined to sleep, should always be kept roused. There is no certain chemical antidote to any of these poisons. Tannic acid precipitates all the alkaloids; hence it has been strongly recommended as an antidote. No injury can follow its exhibition: and a decoction of black tea will be a good substitute for oak-bark or galls. With respect to electricity, Ducros found that the negative current was beneficial to animals poisoned by strychnia or brucia; while the positive current produced convulsions, and accelerated death. (Canstatt, Jahresbericht, 1844, v, 297.) M. Bouchardat advises the antidotal use of a solution of iodine in iodide of potassium: the proportions being three grains of iodine, and six grains of iodide of potassium, to sixteen ounces of water. Half a wine-glassful of this solution should be administered occasionally; and vomiting promoted to eject the compound formed. This antidote is supposed to operate by forming an insoluble compound with the alkaloid, except in the case of digitalia. (Bouchardat, Annuaire de Thérapeutique, 1847, p. 301.) Dr. Garrod recommends, as an efficacious remedy in the early stage of this form of poisoning, the free use of animal charcoal. (Med. Times and Gaz. Dec. 5, 1857, p. 590.) There is no doubt that animal charcoal has a tendency to remove alkaloids from liquids. It has thus been found to precipitate strychnia, and it is equally efficacious with atropia and aconitina. When, however the poison is already in the blood, this and other suggested antidotes would be of no avail. If the poison has been taken in the form of slices of root, as in the case of aconite, it is not easy to perceive how any antidote could prevent the effects. In a case in which I was consulted, the slices of the roots of aconite were found unchanged in the stomach after death,—the poison had been imbibed from them by the mucous membrane of the stomach. Animal charcoal, however, is so far a safe remedy; it can do no injury, and it may act beneficially by partially precipitating the alkaloid, if dissolved, or by enveloping it and sheating the coats of the stomach from its operation, if contained in the roots, leaves, or seeds of the plant. The removal of the poison by emetics, or the stomach-pump, must, however, be the principal object of treatment.

COMMON OR SPOTTED HEMLOCK—(CONIUM MACULATUM.)

This is a well-known hedge-plant, which grows abundantly in most parts of Great Britain. Its effects on man and animals prove that it possesses active poisonous properties: these reside in the seeds, leaves, and roots, and may be extracted by water. Its energy varies, probably according to season and locality. The effects produced by hemlock have not been uniform; in some instances there have been stupor, coma, and slight convulsions; while in other cases, the action of the poison has been chiefly manifested on the spinal marrow,—*i. e.* it has produced paralysis of the muscular system.

Symptoms and Effects.—In a series of cases quoted by Orfila, several soldiers partook of hemlock in soup. Soon afterwards, they all appeared to be intoxicated. One, who had eaten of the soup rather freely, became in less than two hours senseless; he breathed with difficulty; his pulse was hard, small and slow; surface cold; his face livid, like that of a person who had undergone strangulation. Emetics were administered, with temporary relief, but he became again unconscious, lost the power of speaking, and died in three hours after partaking of the soup. On inspection, the stomach was found half filled with a quantity of pulpy matter, and there were some red spots on the membrane, near the intestinal end. The vessels of the brain were gorged with blood, which was quite liquid. (Op. cit. 4ème edit. ii, 427.) In this case the operation of the poison was chiefly manifested on the brain. Dr. J. H. Bennett met with a case which illustrates the other mode of action. A man ate a large quantity of hemlock-plant, by mistake for parsley. In from fifteen to twenty minutes there was loss of power in the lower extremities; but he apparently suffered no pain. In walking, he staggered as if he was drunk; at length his limbs refused to support him, and he fell. On being raised, his legs dragged after him, and when his arms were lifted, they fell like inert masses, and remained immovable. There was perfect paralysis of the upper and lower extremities within two hours after he had taken the poison. There was loss of power of deglutition, and a partial paralysis of sensation, but no convulsions,—only slight occasional motions of the left leg; the pupils were fixed. Three hours after eating the hemlock, the respiratory movements had ceased. Death took place in three hours and a quarter; it was evidently caused by gradual asphyxia from paralysis of the muscles of respiration; but the intellect was perfectly clear until shortly before death. On inspection, there was slight serous effusion beneath the arachnoid membrane. The substance of the brain was soft; on section there were numerous bloody points, but the organ was otherwise healthy. The lungs were gorged with dark fluid blood; the heart was soft and flabby. The stomach contained a green-colored pulpy mass resembling parsley. The mucous coat was much congested, especially at its greater end. Here there were numerous extravasations of dark blood below the membrane, over a space of about the size of the hand. The intestines were healthy, here and there presenting patches of congestion in the mucous coat. The blood, throughout the body, was fluid and of a dark color. A portion of the green vegetable pulp was identified by Dr. Christison, as part of the leaves of the *Conium maculatum*. Some of the leaves bruised in a mortar, with a solution of potash, gave out the peculiar odor of the alkaloidal principle Conia. (Ed. Med. and S. J. July, 1845, 169.)

Dr. Skinner had an opportunity of observing the effects produced by hemlock on five children, varying from five to eight years of age. (Liverpool Med. Chir. Journal, July, 1858.) The symptoms were dryness, with a feeling of constriction in the throat, headache, disposition to sleep, pupils dilated, pulse small and weak, the impulse of the heart weak, breathing slackened, a general paralysis of all the voluntary, and ultimately of all the involuntary muscles, the power of swallowing being the first, and of breathing the last, to fail. At the time of the general paralysis coming on, there was more or less coma and foaming at the mouth, and death appeared to be the result of coma. In two of the cases, the brain was the first organ affected, and the paralysis of the legs never appeared, as the children ran home, complained of sleepiness, and desired to be put to bed.

In a case which occurred to myself, which was the subject of a trial for murder (Reg. v. Bowyer, Ipswich Summer Assizes, 1848), the child died in one hour after swallowing part of a teacupful of a decoction of hemlock, alleged to have been administered by the mother. The child sipped the decoction, until it lost the power of holding the cup; it became insensible and paralyzed, and died in the chair in a sitting posture. There were no morbid appearances, and no hemlock leaves were found in the body, these having subsided in the cup, and being left in the dregs. The child had been poisoned by the upper stratum of clear liquid. The mother was acquitted for want of proof, the death of the child having taken place in secrecy.

Analysis.—Hemlock is known from most other plants, which resemble it, by its large, round, smooth stem, with dark purple spots. The leaves are of a dark green color, and smooth and shining. Every portion of the plant has a peculiar and disagreeable smell when bruised, resembling cat's urine;—a peculiar odor is brought out when the stem or leaves are rubbed with caustic potash, which has been compared to that of mice. A person may be poisoned by decoction or leaves of hemlock, and no leaves be found in the stomach or bowels (case of Bowyer, supra). In this case the stomach had been emptied, and the contents lost, before it was sent to me. No trace of conia was found. The prisoner first gathered the *Anthriscus sylvestris* by mistake for *Conium maculatum:* but it was proved afterwards that she had gathered the leaves of hemlock. A leaf of each of these plants was copied by photography, and produced as evidence in court.

CONIA.—The alkaloid of hemlock is known under the names of conia, conein, conicine, and conicina. It is a volatile liquid like nicotina. It has a pale yellow color, but is darkened by exposure to air. It consists, like prussic acid and nicotina, of carbon, hydrogen, and nitrogen, without any oxygen. Its specific gravity is 0·89. It has the powerful pungent odor of hemlock, with a bitter and intensely acrid taste: it is irritating to the skin. It is transparent, oily-looking, and floats on water, in which it is not very soluble. It gives a temporary greasy stain to paper, and its vapor is inflammable: it produces white fumes with hydrochloric acid vapor. It is very soluble in alcohol and ether, and combines with diluted acids to form poisonous salts. It exists in all parts of the plant, but an alcoholic extract of the seeds yields the largest quantity. The proportion of conia in the plant probably varies at different seasons of the year,—a fact, which will account for the root having been occasionally eaten with impunity. The common *Extract* of hemlock, which owes its properties to this alkaloid, is liable to vary much in strength, according to the mode in which it has been prepared; when over heated, there is a great loss of conia. The presence of this alkaloid in the extract, may be readily determined by triturating it with caustic potash; if present, it is immediately set free, and may be recognized by its peculiar odor, resembling the smell of mice.

Dr. Christison's experiments prove that conia, whether free or combined, is a potent poison. It produces, according to him, general palsy without insensibility, and with slight occasional twitches only of the limbs of the animal. (Op. cit. 855.) The heart was not affected by the poison, as this organ pulsated, even after other signs of life had ceased. Death appeared to be due to asphyxia, from the general paralysis of the respiratory muscles. A single drop of conia, applied to the eye of a rabbit, killed it in nine minutes; and three drops killed a strong cat in a minute and a half.

The effects of this alkaloid have been more recently observed by Dr. Van Praag. (Beil's Journal für Toxicologie, 1 H. 1856, p. 1.) He found it to accelerate respiration, to produce tetanic spasms, and an incurvated state of the limbs and feet, with emprosthotonos: before the cramps set in, there was a tremulous motion of all parts of the body. The symptoms of general paralysis preceding the spasms were manifested in the unsteady gait, the difficulty of standing, the drooping of the head, and the falling together of the knees. The senses and sensibility of the skin were not materially affected. The pupils were dilated and insensible. In some instances the brain was affected, but only slightly, the stupor or tendency to sleep was but slight, and the animal was easily roused. Vomiting or a disposition to vomit, manifested itself in a few instances. On inspection, the brain and its membranes were found congested, the heart sometimes full, at other times its right cavities were empty. The blood was dark and liquid. The latest experiments performed by Orfila, show, that an animal suddenly becomes powerless and falls; there were very slight convulsive motions in the limbs, without opisthotonos, lasting about a minute; the animal then appeared to be in a collapsed state, and died in five minutes. (Mémoire sur la Conicine, 1851, p. 84, and Ann. d'Hyg. 1851, vol. xlvi.)

Analysis.—*Conia* resembles nicotina and ammonia in its liquidity, volatile reaction, and in some of its chemical properties. It is a liquid of oily consistency, of a pale yellow color, powerfully alkaline, and has, when its vapor is diluted, a smell resembling that of mice, and an acrid bitter taste. It gives a volatile greasy stain to paper, and burns with a yellow flame and thick smoke. 1. It is not colored or affected by nitric, sulphuric, or hydrochloric acid; the last-mentioned acid produces with it, dense white fumes of hydrochlorate of conia, and on heating the mixture, this salt remains in prismatic crystals. 2. It is not dissolved by water, but floats on it in oily globules. 3. It is soluble in alcohol and ether, and this last-mentioned liquid removes it from its aqueous solution. 4. It gives a white precipitate with corrosive sublimate, and a yellow precipitate with arsenio-nitrate of silver. 5. It precipitates brown oxide of silver from the nitrate; this is not dissolved by an excess, but the oxide is blackened and reduced. 6. Iodine water gives a reddish-brown precipitate, which is redissolved; an excess of iodine water causes a yellowish precipitate. 7. It gives a yellow crystalline precipitate with chloride of gold, but no precipitate with chloride of platina. 8. Tannic acid precipitates it of a dingy white. 9. Gallic acid gives no precipitate, but slowly acquires a yellowish color. Its odor and solubility in water, as well as several of the characters above mentioned, serve to distinguish it from nicotina and ammonia. It discharges the color of a solution of permanganate of potash more rapidly than ammonia, but more slowly than nicotina.

Organic mixtures.—The process described for nicotina at page 661, will be found effectual for the separation of this alkaloid from the contents of the stomach. It is easily separated by ether from its watery solution; and on the addition of potash, the peculiar odor of conia is at once perceptible.

WATER-HEMLOCK—(CICUTA VIROSA.)

The water-hemlock has given rise to several fatal accidents—its roots having been mistaken for parsnips. The whole of the plant is poisonous; but the roots are the most active, especially when gathered early or late in the year.

Symptoms and Effects.—The symptoms produced by the roots are giddiness, dimness of sight, headache, and difficulty of breathing. There is burning pain in the stomach, with vomiting, and these symptoms are accompanied by heat and dryness of the throat. Convulsions have been observed to precede death. In the cases of three children who died in convulsions from this poison, Mertzdorff found an injected state of the mucous membrane of the stomach, with redness of the air-passages, as well as of the cardia, and pylorus: the vessels of the brain and the sinuses were filled with dark liquid blood. (Wibmer, *Cicuta*, 119.) In a fatal case which occurred to Wepfer, the patient, a man, æt. 20, who had eaten a large quantity of the *root*, was found with his face swollen and his eyes projecting. He breathed with great difficulty, and foamed at the mouth. He was seized with a severe epileptic fit: his limbs assumed a tetanic stiffness, and there was spasmodic breathing. He was quite unconscious, and speedily died. The only marked appearances were fluidity of the blood, and patches of redness on the mucous membrane of the stomach. (Wibmer, loc. cit.)

Dr. Badgley has communicated some cases of poisoning by this plant to the Montreal Medical Gazette (June, 1844): Four children, between five and seven years of age, ate the roots of water-hemlock by mistake for parsnips. Within half an hour, they were all seized with extreme nausea, burning pain at the pit of the stomach, and colicky pains in the bowels: they all complained, on reaching their homes, of sickness, for which warm milk was administered to them. Efforts to vomit were induced: in one, there was full vomiting, but in the other three nothing was ejected from the stomach. The pains gradually increased in two of them; and, in the space of about two hours from the time of their eating the roots, they were laboring under complete coma, with tetanic convulsions,—the jaws rigidly fixed, profound stertor, and the whole face puffed and bloated, having precisely the appearance of the head of a person who had been for some hours under water; pulse intermitting, sometimes imperceptible. Emetics were exhibited, but without effect; and injections of castor-oil and oil of turpentine were employed with great relief. The child who had eaten most sparingly had taken warm milk, and had vomited freely. One died in three hours; the others recovered.

A girl, æt. 8, who had eaten this plant, was found lying quite insensible. Her respiration was feeble, and rattling; the pulse soft, small, and scarcely perceptible; the pupils dilated and fixed; the face pallid; limbs flaccid; abdomen distended; and there was general coldness of the surface, with an entire loss of the power of swallowing. Stimulating embrocations and cataplasms were applied, and after some hours the pupils contracted; the body became warm; the breathing easier; but there were involuntary motions of the limbs. There was a slight return of consciousness and the power of speaking, but the difficulty of swallowing continued; and the patient died in about sixteen hours. (Dr. Schlesier in Canstatt's Jahresb. 1844, v, 299.)

Analysis.—There are no means of identifying this plant except by the determination of its botanical characters. It grows abundantly on the borders of ditches, ponds, and streams. Its stem is thick, round, striated, smooth, sparingly branched, and often attains four feet in height. It is of a reddish color at the branching of the umbels. The leaves are large, pinnated, and serrated: they have the taste of parsley. The root, which has a strong disagreeable smell and an acrid taste, is thick, short, hollow, and has numerous fibres at the joints. The nature of the poisonous principle is unknown.

The CICUTA MACULATA is possessed of equally virulent properties. Many fatal cases have occurred in the United States from the root having been eaten by mistake.

HEMLOCK WATER-DROPWORT—(ŒNANTHE CROCATA.)

This umbelliferous plant grows on the banks of rivers, streams, and ditches. It is one of the most poisonous of the order, and it is considered to be one of the most virulent of English vegetable poisons. It is found growing abundantly in various parts of England and in the south of Ireland. Dr. Pickells has collected thirty cases of death from the eating of the root: the quantity taken in one instance did not exceed the top of the finger in size. The symptoms were insensibility, tetanus, delirium, and insanity. Dr. Christison considers that this plant as it grows in Scotland is not poisonous: but there appears to be no doubt, from various recorded cases, that, as it grows in England, Wales, and Ireland, it is endowed with highly noxious properties.

Symptoms and Appearances.—A set of cases of poisoning by the Œnanthe has been communicated to the Medical Gazette (vol. xxxiv, p. 288), by Mr. Bossey, of Woolwich. A number of convicts, while engaged at work, ate the leaves and roots of the œnanthe. In about twenty minutes, one man, without any apparent warning, fell down in strong convulsions, which soon ceased, but left a wild expression on his countenance. Soon afterwards, as many as nine fell into a state of convulsions and insensibility. The face of the man first seized became bloated and livid; there was a bloody foam about the mouth and nostrils; the breathing was stertorous and convulsive; there was great prostration of strength, and insensibility: he died in five minutes after the symptoms had set in. A second died under similar symptoms in a quarter of an hour, although the stomach-pump was used, and some leaves were extracted with the fluids. A third, who had assisted in carrying the two former, was himself seized with convulsions, and died in about an hour; and soon after him, a fourth died, in spite of the most energetic remedial treatment by cold affusion, emetics, stimulants, stimulating frictions, as well as the use of the stomach-pump. Two other cases proved fatal,—the one in nine days, and the other in eleven; and in these two cases there was irritation of the alimentary canal.

On inspecting the bodies of those who died quickly, there was congestion of the cerebral vessels; and, in one instance, a layer of extravasated blood was found beneath the inner membrane (pia mater). In the first case, which proved most quickly fatal, the cerebral vessels were not congested. The pharynx and gullet had a white appearance, and contained some mucus, with portions of the root. The lining membrane of the windpipe and air-tubes was intensely injected with dark blood. The lungs were gorged with fluid blood. The blood in the heart was very black and fluid. The stomach and intestines were externally of a pink color: the cavity of the stomach was lined with a thick viscid mucus, containing portions of the root. The mucous membrane was much corrugated, and the follicles were particularly enlarged. Similar appearances were met with in all. In the two protracted cases, the mucous membrane of the stomach and bowels was softened and thickened. It had a pink color externally, but no red appearance internally. The vessels of the brain were congested. In the others who partook of the roots the symptoms were not so severe. Under the free use of purgatives, a considerable quantity of the root was discharged, and in a few days the men recovered. These cases show that the œnanthe is a powerful poison. It destroys life with even greater rapidity than arsenic, for it here proved fatal to a strong healthy man in less than *one hour*. Chemists have not yet ascertained on what principle its active properties depend, but they appear to reside chiefly in the root.

In March, 1846, Dr. Unger met with the following cases: A woman dug up some roots which she supposed to be parsnips. They were dressed for dinner, as usual, in an earthen pot in which her food was commonly prepared. The woman, as well as the husband and two children, partook of them. Dr. Unger was suddenly called to see them in the evening, and found them apparently laboring under *delirium tremens*. They were in constant motion, talking incessantly, without knowing what they said, and fancying they saw objects which had no existence. They fought with each other, and were occasionally attacked with fits of convulsive laughter. The countenance was pale, the pupil dilated, the look vague, tongue clean, moist, and tremulous; and the pulse, which, owing to the incessant motion, was felt with difficulty, appeared smaller, weaker, and slower than natural. The patients rejected everything that was offered to them, and were obliged to be restrained by force. A neighbor who had eaten a small portion of the roots, suffered from giddiness and general uneasiness; she was, however, perfectly conscious, and refused to take any remedy. Emetics led ultimately to the rejection from the stomach of a large quantity of the undigested root. After this the symptoms abated, and the next morning, with the exception of a sense of weight in the head, they had all recovered. It is remarkable that there was no purging. (Gaz. des Hôpitaux, Sept. 19, 1846.) The *root* is considered to be the most active part of the plant; it is of a yellowish-white color, and not unpleasant to the taste. A very small portion of it, unless speedily ejected from the stomach, will suffice to destroy life. The symptoms have been occasionally delayed in their appearance; but, as in Mr. Bossey's cases, when they have once commenced, they run on to a fatal termination with great rapidity.

Dr. Woodville relates that three men ate, or rather tasted of the root. One was soon afterwards seized with convulsions, and died; two others suffered from nervous symptoms, including lock-jaw, and one of these died; a fourth had dizziness, and he slowly recovered. It is remarkable that there was no vomiting, nor any tendency to vomit. The following set of cases occurred in Ireland: Eight boys ate the plant for water-parsnip. In four or five hours the eldest became suddenly convulsed, and died; and before the next morning four others died. Of the remaining three, one was maniacal for several hours, the other lost his hair and nails, and the third escaped. (Medical Botany, iv, 144.) They who have vomited at an early stage have generally recovered.

In Sept. 1853, four children ate some of the roots of the œnanthe, the quantity taken being equal in size to a man's thumb. This was at 2 P.M. Four hours afterwards, according to the report of Dr. Nevins, one of them, a boy, was perfectly insensible, and his face was livid and turgid. He had previously vomited blood, and bloody mucus oozed from his mouth on admission. There were violent convulsions affecting the flexor muscles. The trunk was powerfully bent forward, the hands clenched even after death, and the jaws were rigidly closed. The respiration was spasmodic. The pupils were at first contracted, but afterwards dilated; they acted very feebly under the stimulus of light. The pulse was almost imperceptible. This state continued until death, which occurred twelve hours after taking the poison. There was no return of consciousness, and the spasmodic contraction of the muscles continued with slight intermissions as long as the boy lived. The other children recovered. One was insensible and convulsed; a third had only abdominal pain, and no cerebral symptoms. The quantity eaten in the latter cases was unknown. (Mr. Kesteven's Quarterly Report on Toxicology, April, 1854, p. 582, from Association Journal of December 2d, 1853.) In April, 1857, two fatal cases occurred at West Boldon, in Durham. Two laborers ate some of the root of the œnanthe. They were found soon afterwards lying insensible and speechless, their faces livid, tongues swollen and protruded, and there were convulsive movements of the teeth, frothy mucus with blood about their mouths, eyes full and projecting, pupils dilated, breathing stertorous and labored, with occasional general convulsions. They both died in *an hour and a half* from the time at which they were first discovered. On *inspection*, it was found that there had been bleeding from the ears; the abdomen was livid and swollen. The stomach contained a gruelly liquid, with some of the partly digested roots; on removing this liquid, the membrane was found congested and softened. The lungs were engorged with dark liquid blood, and the blood contained in the heart was in a similar state. Mr. Boyle, to whom these cases occurred, forwarded to me a portion of the roots, and there was no doubt that they were the roots of the œnanthe crocata.

In February, 1858, some sailors who had been sent ashore from their ship, collected a quantity of the tubers of the œnanthe, and ate them. Those who suffered, were for the most part suddenly seized with symptoms of a violent kind, which came on in from half an hour to an hour. Some of the men who had eaten the roots were enabled to move about and assist in relieving the others up to the time at which they were themselves attacked. The first man seized was found insensible, and his body immovably rigid: he was moaning and breathing stertorously; his countenance was livid, the eyes were fixed and the pupils dilated; a bloody froth escaped from the mouth. There was opisthotonos; the pulse was feeble, and the action of the heart scarcely perceptible; the lower jaw firmly locked, the tongue much injured, and slightly protruding: death took place quietly in about eight minutes. In another case, in spite of violent vomiting, the man was seized with convulsions, and, after a succession of them, died in two hours. The roots had been for some time in his body before emetics were given. On an inspection of the first case, the skin was livid, the stomach empty, but the mucous membrane was highly congested, and there adhered to it a tough viscid mucus. On opening the abdomen, a pungent odor was perceived, compared to that of burnt celery seed. Some portions of the root were found in the lower part of the small intestines.

In addition to the convulsive symptoms above-mentioned, the patients suffered from cramps in the legs, pain in the course of the spine, extending along the crural and sciatic nerves, giddiness, griping, eructations with the flavor of the root, debility, and total loss of appetite. (Dr. Grahame, in Med. Times and Gaz. March 6, 1858, p. 241.)

It is not often that attempts are made to destroy others by the administration of these vegetable poisons; but a case occurred in France, in which a woman attempted to poison her husband by mixing slices of the root of this plant with his soup. His suspicions were excited by the acrid taste of the soup. The woman was tried for the crime, and M. Toulmouche deposed at the trial, that the plant from which the root had been taken, was the œnanthe crocata,—that it was a powerful poison, and might cause death in two or three hours. The prisoner was convicted, and condemned to ten years at the galleys. (Gaz. Méd. Jan. 3, 1846, 18; also, Journ. de Chim. Méd. 1845, 533.)

Analysis.—The œnanthe crocata can be identified only by its botanical characters. The leaves are of a dark green color, with a reddish-colored border. They have no unpleasant odor when rubbed. The plant bears a greater resemblance to celery than most of the other umbelliferæ. Its stem is channelled, round, smooth, and branched, of a yellowish-red color, and growing to the height of two or three feet. The root consists of a series of oblong tubercles with long slender fibres. It is of a yellowish-white color, and not unpleasant to the taste. It is the most active part of the plant. No poisonous alkaloid has yet been separated from the plant. There are no tests for this poison.

FINE-LEAVED WATER-HEMLOCK—(PHELLANDRIUM AQUATICUM.)

This is another umbelliferous plant, which, like the œnanthe, is often popularly called water-parsnip. It grows by the banks of rivers, ditches, and ponds. It is poisonous, but less virulent than the œnanthe.

Analysis.—The poisonous principle is unknown. The plant has a thick, hollow, smooth, jointed stalk, usually about three feet in height; the leaves are very fine, small, and much subdivided. They are of a dark shining green color; the root is thick, tapering, jointed, and sends off numerous long slender fibres.

FOOL'S PARSLEY—(ÆTHUSA CYNAPIUM.)

FOOL'S PARSLEY, OR LESSER HEMLOCK is very common in gardens and hedge-rows. The leaves so closely resemble those of parsley that they have often been gathered for them by mistake.

Symptoms and Appearances.—The following case of poisoning by the Æthusa cynapium is reported in the Medicinisches Jahrbuch. A woman gave to two of her children some soup, in which she had boiled the root of this plant, mistaking it for parsley. They were both seized with severe pain in the abdomen, and the next morning, one of them, a boy, aged eight years, was in a state of perfect unconsciousness, and his jaws were spasmodically fixed. The abdomen was swollen; there was vomiting of bloody mucus, with obstinate purging,—the extremities were cold, and the whole body was convulsed. He died in twenty-four hours. The only appearances met with, were redness of the lining membrane of the gullet and windpipe, with slight congestion of the stomach and duodenum.

That the root of this plant contains a most energetic poison, and that it is capable of producing rapidly fatal effects, is proved by a case reported by Mr. Thomas, in which death took place in an hour. In May, 1845, a girl aged five years, in good health, ate the bulbs of the æthusa by mistake for young turnips. She was suddenly seized with pain in the abdomen, followed by sickness, but no vomiting. She complained of feeling very ill. On trying to eat, she could not swallow. She was incapable of answering questions, and her countenance bore a wild expression. The lower jaw became fixed, so as to prevent anything being introduced into the mouth. She then became insensible, and died in *an hour* from the commencement of the symptoms: so far as could be ascertained, there were no convulsions. A second child, aged three years, shortly after eating the same substance, was attacked with pain in the stomach, sickness, vomiting, and profuse perspiration. She soon recovered, with the exception of suffering severe griping pains without purging, but these disappeared the following day. A third child, of the same age, suffered from similar symptoms. Recovery in the two last cases was due to the plant having been eaten on a full stomach, and to the effect of early and copious vomiting. (Med. Times, Aug. 23, 1845, 408.) Mr. Thomas injected about two ounces of the juice expressed from the recent bulbs into the stomach of a dog through an aperture in the gullet, which he afterwards secured by a ligature. There were violent spasms and urgent attempts to vomit. In most of the animals upon which this experiment was tried, death took place in from one to four hours. The following case occurred to Mr. Stevenson. Two ladies partook of some salad, into which æthusa cynapium had been put by mistake for parsley. They soon experienced a troublesome nausea, with occasional vomiting; oppressive headache, giddiness, and a strong propensity to sleep, at the same that this was prevented by frequent startings and excessive agitation. There was a sensation of pungent heat in the mouth, throat, and stomach, with difficulty of swallowing, thirst, and loss of appetite. There was numbness, with tremors of the limbs. The two patients only slowly recovered from the effects of the poison. (Churchill's Botany.)

Analysis.—It is known from garden parsley by the smell of its leaves when rubbed, which is peculiar, disagreeable, and very different from that possessed by the leaves of parsley. The leaves of Fool's parsley are finer, more acute, decurrent, and of a darker green color. Its flower-stem, which is striated or slightly grooved, is easily known from all other umbelliferous plants by the beard, or three long pendulous leaves of the involucrum under the flower. The flowers are white,—those of the garden parsley of a pale yellow color.

The poisonous properties of this plant are believed to be due to an alkaloid, the chemical characters of which are unknown.

MONKSHOOD—(ACONITUM NAPELLUS.)

This well-known garden plant is in some parts of the country called *Wolfsbane*, and in Ireland *Blue-rocket*. The roots, seeds, and leaves are highly poisonous, owing to the presence of the alkaloid *aconitina*. The root is especially noxious, and when the leaves have fallen off, it appears to possess its greatest virulence. The different parts of this plant have a numbing burning taste, producing after a short time, tingling of the lips and a hot acrid sensation in the throat.

Symptoms and Appearances.—In from a few minutes to an hour after the poison has been taken, the patient complains of numbness and tingling in the mouth and throat, which are parched: there is giddiness, with numbness and tingling in the limbs, a loss of power in the legs, frothing at the mouth, severe pain in the abdomen, followed by vomiting and purging. In some cases the patient is completely paralyzed but retains his consciousness: in others the giddiness is followed by dimness of sight, delirium, and other cerebral symptoms, but not amounting to the complete coma produced by the cerebral or narcotic poisons. The pupils are dilated, the pulse sinks, the skin is cold and livid, and the breathing is difficult. Convulsions are not commonly observed in man, or are they indicated by general tremors, or twitchings of the voluntary muscles. The poison produces convulsions in animals.

Dr. Fleming, who has closely investigated the subject of poisoning by aconite, considers that this poison may cause death: 1, by producing a powerfully sedative impression on the nervous system; 2, by paralyzing the muscles of respiration, and causing asphyxia; and 3, by producing syncope. The last is the most common form of death in man, when the case is protracted for some hours. A dose sufficiently large to produce death by syncope excites, in the first place, numbness and burning heat in the mouth, throat, and stomach: pain in the abdomen, with sickness and vomiting; diminished sensibility of the skin; giddiness; dimness of vision, or complete blindness; ringing in the ears, and occasionally deafness; frothing at the mouth; a sense of constriction in the throat, with sensations of weight and enlargement of various parts of the body, but especially of the face and ears; great muscular weakness, with general trembling; more or less difficulty of breathing, and speechlessness; sinking at the pit of the stomach; pulse small, feeble, irregular, finally imperceptible; extremities and surface of the body cold and clammy; countenance blanched; and the lips bloodless. The individual dies suddenly; the mental faculties are commonly retained to the last, or there is only slight delirium. The case generally proves fatal in from one to eight hours; if it last beyond this period, there is good hope of recovery. The most common appearance on inspection is a general congestion of the venous system. The brain and membranes are gorged: in some instances there is a redness of the mucous membrane of the alimentary canal. (An Inquiry on the Properties of the Aconitum Napellus, 1845, p. 43.)

Leaves.—In the Lancet for June 28, 1856 (p. 715), is the account of a case of a child, between two and three years of age, who died in about twenty hours, after eating some of the fresh leaves of aconite. The first symptoms were severe pain in the abdomen, vomiting, and a contracted state of the pupils; these were followed before death by collapse and coma. The stomach and intestines were found much inflamed, the latter presenting some patches approaching to gangrene. A boy, æt. 14, ate some of the leaves for parsley. In about two hours he complained of a burning sensation in the mouth, throat, and stomach, and vomited freely. Soon after this he fell on the ground in a fit; and seven hours after having taken the poison, he was found lying across the bed with his hands in his pockets, dead. On inspection, the cerebral vessels were filled enormously with dark-colored fluid blood, upwards of a pound of which escaped from the skull and spinal canal. The stomach was empty: there was a deep inflammatory blush over the whole mucous surface, with patches of a darker color. (Med. Chir. Rev. July, 1844, p. 261. See also case in Lancet, June 28, 1856, p. 715.)

Extract.—In the hospital at Bordeaux, *five grains* of fresh extract of aconite were given to three patients. One of them died in three hours. In a quarter of an hour after taking the poison, the patients had tremors of the muscles, and a pricking sensation over their bodies; severe vomiting followed. They became quite unconscious; and on recovering their senses there was confusion of sight, with intense headache; the skin was cold and clammy, the pulse slow and irregular, and the breathing short and hurried. Two of the patients recovered. (Med. Chir. Rev. Oct. 1839, 544.)

Root.—Poisoning by the *root* of aconite is by no means unfrequent. In the autumn, the root is liable to be mistaken for that of horseradish. It has been thus accidentally eaten on several occasions, and has caused death. It produces, when eaten, a sense of tingling and numbness in the lips, with a burning sensation in the mouth and throat, extending to the stomach. (Pereira, Mat. Med. vol. ii, pt. ii, p. 688.) A fatal case arising from the root having been eaten by mistake for horseradish occurred at Bristol in the autumn of 1853. The deceased in this case is stated to have taken only as much as would go on the point of a table knife. Mr. Herapath calculated the quantity at thirty-five grains, and estimated it to be equivalent to one-twentieth of a grain of pure aconitina. A similar mistake led to fatal results in three hours in a case which occurred at Lambeth; and another set of cases occurred at Dingwall, in Scotland, in January, 1856. Here three persons were poisoned by reason of their having had sauce, made with the root of aconite, served at dinner with roast beef in place of horseradish sauce! They were healthy adults; they all died within three hours and a half. Mistakes of this kind show deplorable ignorance, but there is always the risk of their occurrence when horseradish and aconite are grown near to each other in a garden, at that season of the year when the leaves have fallen.

A trial for murder by poisoning with the root of this plant took place at the Monaghan Lent Assizes in 1841 (Reg. v. McConkey), in which Dr. Geoghegan, of Dublin, conducted the medico-legal investigation. The medical evidence was beset with difficulties: for no trace of the poison could be discovered in the body, and it was only by a close analysis of symptoms and appearances, that the charge was brought home to the prisoner. The deceased had eaten for his dinner some greens dressed for him by the prisoner; he complained of their having a sharp taste, and this was perceived also by another person present, who tasted them. It was ascertained that the deceased, soon after the meal, had vomited a greenish matter, and suffered from purging, restlessness, incoherence, lock-jaw, and clenching of the hands. He died in about three hours after having eaten the greens, but was not seen by a medical man while living. The chief appearance met with was in the stomach, where the mucous membrane was of a light reddish-brown color. Traces of vegetable matter were found in the intestines: but no poison could be detected either botanically or chemically. The symptoms suffered by a friend of the deceased, who had accidentally tasted the greens, were very characteristic of poisoning by aconite. In *two* minutes he felt a burning heat in the mouth, throat, gullet, and stomach; then a sensation of swelling in the face, with a general feeling of numbness and creeping of the skin. Restlessness, dimness of sight, and stupor almost amounting to insensibility, followed; and in about an hour after the meal, he was found speechless,—frothing at the nose and mouth, the hands and jaws clenched, appearing occasionally as if dead, and then again reviving. Vomiting, purging, tenderness at the pit of the stomach, cramps, tingling of the flesh, and a burning taste in the mouth followed. This man did not entirely recover until after the lapse of five weeks. The prisoner was convicted of murder, and confessed before her execution that the powdered *root* of aconite had been mixed with pepper and sprinkled over the greens. (Dublin Med. Journal, vol. xix, p. 403.)

This case, decided in 1841, bears some analogy to that of Palmer, which occurred fifteen years later. It establishes the truth of the proposition that a conviction for murder by poison may take place in the absence of any chemical evidence of the nature of poison. The non-discovery of the vegetable or of the alkaloid aconitina in the body was raised as an objection to the opinion of Dr. Geoghegan in the case of McConkey; but the medical and general evidence taken alone was considered to be conclusive of the fact of poisoning.

Dr. Geoghegan, in the paper referred to, quotes two other instances of poisoning by aconite, one of a man, aged fifty-six, who died in an hour and a quarter after eating the root; and the second, a boy aged seven, who died in two hours, having been much convulsed before death.

It is stated that one drachm of the root has been known to prove fatal; but it is probable that less than this would cause death. In November, 1856, Mr. Hadfield forwarded to me four small slices of the root, taken from the stomach of a man who died in three hours. The quantity which he had swallowed with suicidal intention was unknown; but none was thrown off by vomiting, so far as could be ascertained. The *symptoms* within half an hour of death were burning pain in the stomach, parched mouth,—intense thirst,—retching and vomiting of a tenacious mucus,—cold perspiring skin,—imperceptible pulse, and a feeling of deadly sickness. The patient was conscious: there were no convulsions. On *inspection*, there was congestion of the brain as well as of its membranes; the heart was flaccid: it contained some blood on the right side. The stomach contained much half-digested food, with four slices of aconite root apparently unaltered. The mucous membrane presented a slight reddish-brown patch at the greater end, of the size of half a crown. It was otherwise healthy as well as the other organs. (For an account of poisoning by this plant I must refer the reader to a paper by Dr. Geoghegan, Dub. Jour. Med. Sci. vol. xix, p. 403.)

Tincture.—There are numerous instances recorded of poisoning by aconite under the form of tincture of the root. In a case which occurred to M. Devay (Cormack's Edinburgh Journal, April, 1844), a man is stated to have recovered in three days after having taken upwards of ten drachms of the tincture (only infused for a day); but this could have contained no aconitina. The late Dr. Male, of Birmingham, died from the effects of not more than *eighty drops* taken in ten doses, over a period of four days,—the largest quantity taken at once being *ten* drops. (Prov. Med. and Surg. Journ. August 20, 1845, p. 535; also Med. Gaz. xxxvi, p. 861.) The late Dr. Pereira informed me that he had known tingling and general numbness of the limbs produced in hysterical females by a dose of only *five minims* of a carefully prepared tincture. Dr. Topham has published an account of the symptoms produced by *fifteen minims* of the tincture of the root of aconite. Immediately after taking the poison in a mixture into which it was put by mistake, the patient (a woman, æt. 27) felt a sensation of numbness in the tongue, with difficulty of swallowing. There were convulsive twitchings of the muscles of the face, and she lost the power of walking. There was complete unconsciousness, which continued for two hours, when she began to recover. The pupils were observed to be slightly contracted. The intensity of the symptoms varied at intervals, and came on in paroxysms. They indicated great disorder of the nervous system. The next day she had numbness in both arms, but she rapidly and perfectly recovered. (Lancet, July 19, 1851, p. 56.)

In January, 1853, a case of poisoning by tincture of aconite occurred at a convent near Bristol. One of the inmates named "Forty" had administered to her, by mistake, *seventy minims* of *Fleming's tincture* of the root mixed with one grain of acetate of morphia. This was about seven o'clock in the morning. In a few minutes she became very thirsty, complained of a burning sensation and pain in her stomach, to relieve which she swallowed a quantity of cold water. In fifteen minutes there was violent vomiting, which continued for two hours. She lost the power of standing, and was very restless. The pain in the stomach increased. After the first hour she was unable to do more than turn her head and vomit. There was violent straining as well as convulsive movements of the muscles. At nine o'clock she had a stupefied look, complained of giddiness, and was covered with a cold sweat. At ten o'clock she was quiet, as if asleep. She was conscious until shortly before her death, which took place in about four hours after she had taken the poison. There were no general convulsions: the pain in the stomach was well marked throughout. On *inspection*, the face and lips were found swollen and dark-colored, eyes bright, pupils dilated, and the muscular system rigid. The membranes of the brain were congested, but the brain itself was firm and healthy. The lungs were healthy: there was merely cadaveric congestion from gravitation. The heart was flaccid, uterus congested, bladder empty, and sphincter ani relaxed. The stomach contained some mucus, and the membrane at the larger curvature was injected (reddened) in patches, but otherwise natural. The mucous membrane of the duodenum was in a high state of inflammation, abraded in patches, softened, and broken down. Some spots were of a very dark color, passing to mortification. It is proper to observe that the deceased died on the 5th of January, and the inspection was not made until the 14th. (Report by Dr. O'Bryen, Association Med. Jour. Jan. 28, 1853, p. 92. See also a case in the Lancet, 1855, vol. i, p. 467.) In this case a female died in five hours from two drachms of the tincture taken with suicidal intention. There were narcotic symptoms.

In 1853, a healthy young man lost his life at Glasgow, by reason of his having taken a mixture containing *twenty-five minims* of tincture of aconite, twenty minims of tincture of belladonna, and a drachm of the tincture of musk. The tincture in this case was prepared with sixteen ounces of the root of aconite to thirty fluid ounces of spirit. The mixture was swallowed at 6.30; the patient walked to a friend's house about three-quarters of a mile distant, which he reached at 7.20. He then complained of being sick, and of a tingling sensation in his hands and arms. In a short time his hands and arms were so benumbed and powerless, that when he raised them he could not keep them up. Vomiting came on, with convulsive movements of the body, the pulse could not be felt, and the patient, retaining his consciousness to the last, died within three hours from the time of taking the poison. The body was inspected two days after death by Dr. Easton. The veins of the brain were unusually congested, and there was a great quantity of serum effused in the arachnoid (membranes). The lungs and the right cavities of the heart were gorged with a dark blood. The lining membrane of the stomach was of a dark red color. Death was very properly referred by Dr. Easton to the action of aconite. (Assoc. Med. Jour. Sept. 16, 1853, p. 817.) In October, 1852, an excise officer lost his life by merely tasting Fleming's tincture of aconite, under the supposition that it was flavored spirit. He was able to walk from the Custom House over London Bridge, but he died in about four hours after taking the poison.

Several cases of poisoning occurred some years since at Lille, in which tincture of the fresh root of aconite was taken by mistake for a cordial. The symptoms appeared in three members of a family in half an hour: there was severe burning pain in the throat and stomach, with vomiting, purging, and tenderness of the abdomen. One died in two hours; the second in two and a half hours; the third, who had delirium, recovered. The only appearance met with on inspection, was great redness of the mucous membrane of the stomach and small intestines. (Ed. M. and S. J. xxviii, 452.)

In February, 1856, Paymaster Kent died from the effects of one drachm of the tincture, taken by mistake. The symptoms were giddiness with intense burning pain from the tongue to the throat, a loss of power in the legs, coldness of the hands and feet and along the spine, twitchings of the muscles of the face, fingers, and toes, dimness of vision, dilatation of the pupils, inability to swallow, weak pulse, and involuntary evacuations. Just before death there was one universal convulsion. He revived, gasped, and then died. His skin at this time was cold, his lips were blue, his pulse was scarcely perceptible, and his breathing oppressed. He died an hour and a half after he had taken the poison, which had been dispensed by mistake for another tincture. Emetics produced active vomiting, but the poison had become absorbed. On inspection, the stomach contained a dark-brown fluid; the mucous membrane was congested in patches of various sizes, and of a dark-red color. The right side of the heart, which was healthy, was filled with liquid venous blood. The liver, spleen, kidneys, and intestines were congested. The bladder was empty. (Dr. Bone, in Lancet, April 5, 1856, p. 369.) In September, 1857, the wife of a physician, of Durham, died from the effects of one drachm of tincture of aconite given in two doses, at an interval of some hours. It had been given by mistake for tincture of henbane.

The tincture of aconite, according to the London and Dublin Pharmacopœias, is made by infusing the *root* in rectified spirit. Fleming's tincture is also made with the root, but with half the quantity of spirit. The *medicinal dose* is variously stated, owing to the great difference in the strength of this preparation. It should not exceed five minims. The late Dr. Pereira states that a dose of six minims administered twice produced the most alarming symptoms in a healthy young man. (Mat. Med. vol. ii, pt. ii, p. 693.) Fleming's tincture is a powerful preparation, and might, from its appearance, be mistaken for sherry wine. Since this tincture is as deadly in its operation as prussic acid, and so many accidents have occurred from the use of it, it seems advisable that its strength should be reduced.

A well-marked case of poisoning by a *decoction* of this plant occurred to Mr. Sayle. A man, æt. 39, boiled the fresh stalks and leaves of aconite in half a pint of beer until it was reduced to a quarter of a pint: he then swallowed half of it as a medicine. An hour afterwards he was found in bed, rolling his arms about and foaming at the mouth: the pupils were widely dilated, the legs were paralyzed, the skin was cold and clammy, there was great nausea, the pulse was scarcely perceptible, and he was perfectly insensible. He soon afterwards died. The abdomen was examined, and the only appearance met with was a slight redness near the cardiac extremity of the stomach. (Med. Times, Oct. 18, 1845, p. 70.)

Analysis.—The botanical characters of the leaves and root, when any portions can be obtained, will enable a medical witness to identify this vegetable poison. The root has been frequently and fatally mistaken for horseradish, but there are these striking differences: 1. Aconite phocyanide of potassium produced no change in the solution. In *organic liquids* the alkaloid is sufficiently soluble in ether to be separated by the process of Stas (see page 693). Dr. Headland has recommended as a physiological test for the presence of this alkaloid, the application of a spirituous extract of the acid contents of the stomach. If 1-20th of a grain be obtained it will be sufficient. He states that 1-300th of a grain will poison a mouse with characteristic symptoms; 1-100th a small bird; 1-1000th of a grain causes tingling and numbness of the tip of the tongue; 1-100th dissolved in spirit and rubbed into the skin causes loss of feeling, lasting for some time. (Lancet, March 29, 1856, p. 343.)

DEADLY NIGHTSHADE—(ATROPA BELLADONNA.)

There are several plants known under the name of Nightshade, which however, differ much from each other. The **Woody Nightshade (Solanum Dulcamara)**, and the **Garden Nightshade**, or **Solanum Nigrum**, known by the red and black color of their berries, have been elsewhere described (page 670). The vegetable poison now to be described is the **Deadly Nightshade**. The leaves, berries, and root of the plant are poisonous. They owe their noxious effects to the presence of the alkaloid *Atropia*.

Symptoms.—The symptoms produced by this poison are of a uniform character, and as a summary they may be thus described: Heat and dryness of the mouth and throat, nausea—vomiting—giddiness—indistinct or double vision—delirium,—great excitement,—convulsions—followed by stupor and lethargy. The pupils are much dilated and the eyes are insensible to light. In two cases which occurred to Mr. Tufnell, the pupils were contracted during sleep, although dilated in the waking state. (Dub. Med. Press, Jan. 5, 1853. Journal de Chimie Medicale, 1853, p. 695.) Several deaths from the effects of the *berries* occurred in this metropolis in the autumn of 1846. The following case was admitted into Guy's Hospital. A boy æt. 14, ate, soon after breakfast, about thirty of the *berries* of belladonna, which he had bought in the street. In about three hours it appeared to him as if his face was swollen,—his throat became hot and dry,—vision impaired,—objects appeared double, and they seemed to revolve and run backwards. His hands and face were flushed, and his eyelids swollen; there were occasional flashes of light before his eyes. He tried to eat, but could not swallow on account of the state of his throat. In endeavoring to walk home he stumbled and staggered; and he felt giddy whenever he attempted to raise his head. His parents thought him intoxicated; he was incoherent,—frequently counted his money, and did not know the silver from the copper coin. His eyes had a fixed, brilliant, and dazzling gaze; he could neither hear nor speak plainly, and there was great thirst; he caught at imaginary objects in the air, and seemed to have lost all knowledge of distance. His fingers were in constant motion: there was headache, but neither vomiting nor purging.

He did not reach the hospital until nine hours had elapsed; and the symptoms were then much the same as those above described. He attempted to get out of bed with a reeling, drunken motion: his speech was thick and indistinct. The pupils were so strongly dilated that there was merely a ring of iris, and the eyes were quite insensible to light. The eyelids did not close when the hand was passed suddenly before them. He had evidently lost the power of vision; although he stared fixedly at objects as if he saw them. The nerves of common sensation were unaffected. When placed on his legs he could not stand. The pulse was 90, feeble, and compressible: his mouth was in constant motion, as if he were eating something. His bladder was full of urine on admission. He continued in this state for two days, being occasionally conscious; when, by a free evacuation of the bowels, some small seeds were passed: these were examined and identified as seeds of belladonna. The boy gradually recovered, and left the hospital on the sixth day after his admission: the progress of recovery was indicated by the state of the pupils, which had then only acquired their natural size and power of contraction. In three other cases which occurred at the same time, the berries having been baked in a pie, pains in the limbs, drowsiness, insensibility, and convulsions, were among the symptoms. In two cases of poisoning by the berries, related by Dr. Moll, the symptoms bore a strong resemblance to those of delirium tremens, but among them were heat and dryness of the throat, loss of power of swallowing, incoherent speech, double vision, and strange spectral illusions, with occasional fits of wild and ungovernable laughter. On the following morning both these patients recovered as if from a dream; but they suffered for some time from languor, thirst, and dryness of the throat: the pupils also continued dilated. (Casper's Wochenschrift, 10 Januar, 1846, p. 26.) Two cases of the effects of the berries on children are quoted in the Edinburgh Medical and Surgical Journal. (Vol. xxix, p. 452.) Among the first symptoms, three hours after the berries were eaten, the children were seized with uncontrollable fits of laughter; catching at objects; incessant, incoherent babble, and continual agitation of the body, with fixed staring eyes, and dilated insensible pupils. A man, æt. 34, ate about fifty berries to relieve his thirst. He soon perceived a burning sensation in the throat, and a feeling of stupefaction. He staggered home and went to bed. In the evening he was seized with such violent delirium that it required three men to confine him. His face was livid; his eyes were injected and protruding,—the pupils strongly dilated; the carotid arteries pulsated most violently; and there was a full, hard, and frequent pulse, with loss of power to swallow. He was bled, and in about half an hour was able to swallow an emetic: this brought away a violet-blue or purple liquid, which is always a well-marked indication of this form of poisoning. Purgative medicines and injections were employed, and the man recovered his consciousness in about twelve hours. (Case by Dr. Rosenberger, Canstatt's Jahresb. 1844, v, 295.) In six other cases, reported in the same journal by Dr. Teschenmacher, the symptoms varied slightly in the different patients. They all experienced double vision, dilatation of the pupils, constriction of the throat, giddiness, and a tendency to sleep. They who had eaten most berries fell into a soporose state, and had violent convulsions of the extremities. In twenty-four hours the whole of the family had recovered. (Ib. p. 296.)

The *root* of the belladonna, administered in the form of decoction as a clyster, has destroyed life. Four scruples of the root were employed, and the liquid, strained and reduced by evaporation to four ounces, was injected. After a slight stage of excitement, the patient, a female, æt. 27, fell into a state of complete coma; the countenance appeared swollen, and of a reddish-brown color; the pupils were excessively dilated; the pulse was at first full and hard, then small; death took place in five hours. (Casper's Wochens. 8 Feb. 1845, p. 101.) This case proves that, in poisoning by nightshade, there is in some instances little or no delirium, and that the patient may be at once thrown into a fatal lethargy. A case of recovery, in which a girl, æt. 9, masticated portions of the *root* of belladonna, is reported by Mr. Bullock. (Med. Gaz. xix, 265.) In two hours there was sickness, lassitude, and dryness of the throat; in four hours delirium, with convulsions, came on; the face was distorted, eyes protruded, and pupils widely dilated. The girl was completely insensible. Under the use of the stomach-pump and emetics she recovered.

The *leaves* of belladonna have occasionally given rise to accidents. A young man swallowed an infusion of two drachms of the leaves. In about an hour he found great difficulty in swallowing, the salivary secretion was suppressed, and objects appeared to be in perpetual motion before him. He became delirious, attempted repeatedly to pass his urine but could not; and for an hour and a half he was in constant motion, although his gait was unsteady. The muscles of his face, jaws, and limbs were agitated by convulsive twitchings: the pupils were excessively dilated, and there were singular hallucinations. There was neither nausea, vomiting, nor purging. Emetics, injections, and bleeding were resorted to, and the next morning he awoke as if from a dream. (Ann. d'Hyg. Oct. 1847, p. 413.)

Dr. Garrod has communicated to me the symptoms which one of his patients and himself suffered as a result of taking an infusion of belladonna leaves, which had been ignorantly supplied for ash-leaves. A quarter of an ounce of the leaves was boiled for a few minutes with ten ounces of water. Dr. Garrod took about half a wine-glassful of this decoction, equivalent to eleven grains of dried leaves. He believed, at the time, it was the infusion of ash-leaves, and wished to determine by taste, whether it was good. In about half an hour, the symptoms commenced by swimming in the head,—intense feeling of nervousness,—palpitation of the heart,—a small and rapid pulse,—dryness of the mouth and throat, and perversion of taste,—indistinctness of vision,—dilatation of the pupils,—rapid flow of ideas, weakness of the limbs, and slight difficulty of articulation. His taste was so altered that some brandy given to him had the taste of the infusion. Some of the infusion was put into an eye, and in about a quarter of an hour it dilated the pupil powerfully. In about four hours, Dr. Garrod had recovered from these effects: but there was indistinctness of vision with dilated pupils for one or two days, —and a generally depressed state of the nervous system, from which even after two years, he had not entirely recovered. His patient took half a pint of the infusion equivalent to about a quarter of an ounce of dried belladonna leaves; the symptoms were similar but more severe, and lasted for a longer time. They were giddiness, difficulty in walking,

—dryness of the mouth and throat, and perversion of taste,—indistinctness of vision, and dilatation of the pupils,—bloodshot eyes,—difficult articulation,—delirium,—coma (insensibility),—scarlet redness of the skin of the face and neck, followed on the second day by a peeling off of the cuticle. This last symptom, although not common, has been observed in other cases of poisoning by belladonna in large medicinal doses. Some of these symptoms continued more or less for ten hours. The patient suffered from shock to the nervous system for a considerable period after his recovery from the urgent symptoms. One of the effects of the poison was to produce, in both cases, an increased secretion from the kidneys.

The *extract* of belladonna appears to be very uncertain in its operation. The medicinal dose is from one to five grains. In a case which occurred at St. George's Hospital, an ounce of the extract was taken without causing death; but, in another instance, a child, æt. 9, nearly lost his life by a dose of thirty grains, administered to him in mistake for extract of taraxacum. Delirium came on in half an hour; this was followed by coma. In addition to other characteristic symptoms, the child suffered from convulsive twitchings of the arms. There was pain in the head, with deranged vision for ten days after the accident. (Prov. Med. Jour. Feb. 24, 1847, 98; see, also, Pharmaceutical Journal, Feb. 1853, p. 404.) Dr. Gray, of New York, has related a case, in which a child, between two and three years of age, swallowed from eight to twelve grains of the extract, and after suffering the usual symptoms in a severe form for three hours, recovered. This gentleman describes his own sensations after having taken a large dose of the same preparation. They bear out singularly the truth of the descriptions given by other observers. (See Med. Gaz. xxxvii, 255.) Mr. Iliff, Jr., has given an account of the effects produced on himself by a dose of nine grains of the extract of belladonna, for which I must refer the reader to the Lancet (Dec. 1, 1849, p. 756; see, also, Med. Times, Aug. 30, p. 234, and Ann. d'Hyg. 1853, i, 417). In the latter case, the members of a family were poisoned by the extract, but they all recovered. In the Medical Gazette (vol. xlii, p. 589), will be found the report of an inquest in a case of alleged poisoning by belladonna, involving many points regarding this poison. The question at issue was, whether death had arisen from an overdose of the extract, or from natural causes. The extract of belladonna is subject to great variation in strength,—a fact, which may furnish an explanation of certain exceptional cases in which persons have recovered from large doses of this compound. A case occurred to Mr. Edwards, in which a female, æt. 34, recovered after having swallowed a drachm of the extract by mistake. (Lancet, May 24, 1851, p. 568.) Mr. Solly met with an instance, in which a man took a scruple by mistake. No symptoms occurred for two hours. He then suffered from dryness of the throat, difficulty of swallowing, fanciful delusions, and rambling, incoherent conversation. The pupils were dilated and insensible to light, —the eyes were prominent and had a vacant stare. There was drowsiness with a feeble and irregular pulse, and a loss of muscular power. Under the use of emetics, the man recovered the next day. (Lancet, Feb. 3, 1855, p. 121.)

Two persons swallowed a small spoonful of the extract of belladonna by mistake for that of juniper. There was speedily indistinctness of vision, tottering gait, delirium, incoherency, hallucination, and dilatation of the pupils. In one there was great cerebral excitement. The apothecary, to whom the extract was taken, tasted it, and soon experienced symptoms which led to a suspicion of its real nature. Under treatment, the symptoms of poisoning disappeared in two days: but one of the patients died on the seventh day from disease. The physical and physiological properties of the extract indicated that it was belladonna: but the attempt to procure atropia entirely failed. A portion of the concentrated extract given to a dog caused dilatation of the pupil in a quarter of an hour, an index of the rapidity with which the alkaloid atropia is absorbed and diffused through the blood. A woman swallowed on an empty stomach a drachm of the extract of belladonna. She then took some food. After the lapse of three hours, symptoms came on suddenly. She lost the power of standing; there was trembling of the limbs with convulsive motions,—a nervous laugh and incoherent speech. The pupils were much dilated, and great lassitude followed this stage of excitement. Under treatment, she recovered in twelve hours. (Ann. d'Hyg. Oct. 1847, p. 413.) Orfila has satisfactorily accounted for these anomalies in the power of the extract. Some specimens are quite inert: those only have an energetic action which are prepared by evaporating the fresh juice at a very low temperature. (Toxicologie, ii, 395.)

Local action.—The extract, as it is well known by its effects in dilating the pupil, acts through the *skin*. It is easily absorbed, and must therefore be used with caution. M. Casanova ordered a blister to be applied to the abdomen of a female, and prescribed a dressing of one part of extract of belladonna to three parts of mercurial ointment. At first, nine grains, and after two hours, thirty grains of the extract were thus employed. The patient was soon attacked with most violent delirium, crying out incoherently, and attempting to drive away horrible forms which she fancied she saw flitting around her. The pupil was enormously dilated; there was intense thirst with spasmodic constriction of the throat in drinking. These symptoms did not disappear until after the lapse of forty-eight hours. (Gaz. Méd. 13 Mars, 1847, 207.) In a case that occurred to Dr. Jenner, symptoms of poisoning arose from the application of a fresh belladonna plaster to a pustular surface produced by the application of an old plaster. Some time afterwards the patient suffered from great dryness of the tongue and throat, which prevented distinct articulation, and was rather increased by his taking water. There was a great desire to pass the urine, but only a few drops could be passed at a time. There was confusion in the head, and convulsive catchings in the limbs. In about eight hours he had lost the power of standing. He was restless,—his hands were in constant motion, as if he were busy in moving light objects. He moved his mouth incessantly, but the sounds thus made were unintelligible. He seemed unconscious of the presence of persons. The pupils were large and they acted imperfectly under exposure to light. On the removal of the plaster the symptoms ceased, leaving the next day dilatation of the pupils, dimness of vision, and impairment of memory. (Med. Times and Gazette, Nov. 22, 1856, p. 513.)

Appearances.—The appearances observed in several cases of poisoning by the berries, which proved fatal in London during the autumn of 1846, were as follows: the vessels of the brain were congested with liquid blood; the stomach and intestines were pale and flaccid; there were some red spots towards the cardiac end. In other fatal cases, of which the appearances have been reported, the vessels of the brain and its membranes were found distended with thick black blood. Red spots have also been observed around the throat and gullet, and congested patches of a dark purple color on the coats of the stomach. In some instances the mucous membrane had been completely dyed by the juice of the berries. A boy, æt. 5, after having eaten a quantity of the berries of the belladonna, went to bed, was very restless, vomited once, and died in convulsions about fifteen hours after having taken the poison. On inspection, the eyes were half open, with an intense lustre; the pupils dilated; the mouth spasmodically closed, and the sphincter ani relaxed. The cerebral vessels were distended with dark-colored blood; the substance of the brain, cerebellum, and medulla oblongata, presented numerous bloody points. In the throat and gullet there were several patches of redness. In the stomach there was some fluid with three open berries; the mucous membrane was of a reddish-blue color in various parts. (Case by Dr. Rosenberger, Canstatt's Jahresb. 1844, v, 295.)

Analysis.—The indigestible nature of the leaves, fruit, and seeds will commonly lead to their detection in the matters vomited or passed by the bowels, or in the contents of the viscera after death. The seeds of belladonna are very small, and areolated on the suface; they cannot easily be distinguished from those of hyoscyamus. The coloring matter of the berry is of a deep-purple hue; it is turned green by alkalies, and red by acids. The leaves would be known by their botanical characters, and a decoction or infusion of them by the liquid causing dilatation of the pupil. Dr. Runge states that the urine, blood, or organic liquids containing this poison, applied to the eye of an animal, cause dilatation of the pupil. Orfila has not observed this effect in poisoning by belladonna (Op. cit. ii, 267), and even if it occurred, he considers that it would be too vague a sign for diagnosis, as it takes place equally with henbane and stramonium.

Atropia.—Atropia is the name given to the alkaloidal principle of belladonna. This alkaloid is a powerful poison. In November, 1850, Mr. Sells, of Guildford, forwarded to me for examination the stomach of a young man who had poisoned himself by taking *two grains* of atropia. He took the dose on going to bed. He was heard to snore heavily during the night, and was found dead about seven o'clock in the morning, lying on his right side, the surface livid, the limbs rigid and contracted, and with a little brown matter issuing from the mouth. The pupils were much dilated. The mucous membrane of the stomach presented a diffused redness, which might have arisen from some brandy which he had swallowed. No trace of the poison could be detected in the stomach or its contents. In the Association Medical Journal (Sept. 16, 1853, p. 818) will be found the report of a case in which all the symptoms of poisoning by belladonna arose from the application of a weak solution of atropia and water to the conjunctiva.

Analysis.—Atropia is a white crystalline substance, requiring 500 parts of water to dissolve it: it is easily dissolved by alcohol, ether, and diluted acids. It forms crystallizable salts. It melts at a low temperature, and burns with a bright smoky flame. 1. Iodine water gives with its salts a dense brown precipitate. 2. Tannic acid precipitates it of a dingy white. 3. Gallic acid has no effect. 4. Nitric and sulphuric acids dissolve it, but do not produce any change of color. Sulphuric acid and bichromate of potash produce with it green oxide of chromium. 5. It is rendered turbid by chloride of platina, and is precipitated of a yellowish-white color by chloride of gold. In *organic liquids* its presence, if in sufficient quantity, may be detected by the process of Stas (p. 698). A solution of any of the salts applied to the eye produces dilatation of the pupil.

INDIAN TOBACCO—(LOBELIA INFLATA).

The powdered leaves of Indian tobacco contain an acrid principle which is capable of producing poisonous effects on the brain and spinal marrow attended with irritation of the stomach and bowels. As a poison it has only recently become known in this country. Wibmer relates that in one instance it produced at first violent vomiting in the person for whom it was prescribed; but the medicine was repeated until it was no longer ejected from the stomach. The patient suffered severe pain, and speedily died: stupor and convulsions having preceded death. The powdered leaves and seeds are much employed by quacks in the United States; and accidents occasionally arise from the substance being prescribed in excessive doses. When administered in doses of from ten to twenty grains, lobelia acts as an emetic; but in larger quantity it acts deleteriously. It would also appear that even ordinary medicinal doses affect some individuals with great severity. There is an erroneous notion that this is a useful medicine and not a poison, although it may be either, according to the mode in which it is employed.

In one case a man lost his life by swallowing *one drachm* of the powdered leaves, prescribed by a quack. This person was seen by a medical practitioner soon after he had taken the poison; he was evidently suffering great pain, but he was quite unconscious,—the pulse was small, the pupils were strongly contracted, and insensible to light. He had vomited the greater part of the poison. He suffered from spasmodic twitchings of the face, sank into a state of complete insensibility, and died in about thirty-six hours. On inspection, some fluid was found in the stomach, but none of the powder. The mucous membrane was intensely inflamed, and the vessels of the brain were strongly congested. (Pharm. Times, May 1, 1847, p. 182.)

The *seeds* of lobelia are equally poisonous. In the Med. Times and Gazette, Nov. 26, 1853, p. 568, two cases are reported in which the seeds proved fatal. In one, the mucous membrane of the stomach was highly inflamed. Another case is referred to in the same journal, March 12, 1853, p. 270.

Within the last few years, there have been several inquests and trials for manslaughter in this country as the result of the improper administration of the leaves of the *Lobelia inflata* by ignorant quacks, calling themselves medical botanists and dealers in vegetable medicines. The medical evidence given on these trials showed that in large doses lobelia is a most noxious drug. (See Medical Gazette, vol. xliv, pp. 383 and 433; vol. xlvi, p. 384; Lancet, March 5, 1853, p. 237; Pharm. Jour. Aug. 1851, p. 87; and for some remarks on the action of the poison, see a paper by Mr. Curtis and Dr. Pearson, Med. Gaz. 1850, vol. xlvi, p. 285; also Pereira, Mat. Med. vol. ii, pt. ii, p. 12.) Those impostors who profit by the prescription and sale of this drug among the ignorant poor, maintain the doctrine that it cannot kill, and never has been known to destroy life! From inquiries recently made, I have reason to believe that deaths from lobelia still frequently occur in the northern counties. The noxious medicine is taken in secrecy, under the direction of quacks, and any death arising from it is either concealed or referred to other causes. The late Sergeant Wilkins was a great and successful defender of this class of poisoners: out of several trials for manslaughter, in which the cause of death, medically speaking, was clear, he succeeded in winning a favorable verdict from the juries. In July, 1856, one of these quacks was, however, convicted on a charge of manslaughter, for killing a woman with overdoses of lobelia. Severe pain, followed by loss of consciousness, and congestion of the brain, were the chief symptoms preceding death in this case. The admission that, in proper doses, it was a useful remedy in spasmodic asthma was of no avail on this occasion. The man was sentenced to three months' imprisonment. (Reg. v. Boyden or Jackson, Lincoln Summer Assizes, 1856.) A man named Riley Drake was convicted in the United States of having caused the death of a woman by administering lobelia in improper doses. (Wharton and Stille's Med. Jur. p. 522.)

Analysis.—Lobelia is seen in the form of a greenish-colored powder (fragment of leaves). This powder acquires a reddish-brown color from strong nitric acid, and is blackened by concentrated sulphuric acid. Iodine water has no effect upon the infusion. The proto- and persulphate of iron produce with it a dark-green color, the persulphate, very rapidly. Lobelia can be safely identified only by the botanical characters of its leaves and seeds. The leaves and seeds contain a resinoid substance called *Lobelin*, which has the smell and taste of the plant. It acts as a powerful emetic in doses of from one half to one grain.

FOXGLOVE—(DIGITALIS PURPUREA).

Purple foxglove is a well-known hedge-plant growing abundantly in the southern districts of England. All parts of the plant,—the seeds, leaves and root are poisonous, owing to the presence of the poisonous alkaloid—*digitalia.* The leaves, whether in the form of powder, infusion, extract, or tincture, exert an action on the brain and spinal marrow as well as on the stomach and bowels. They retain their noxious properties when dried.

Symptoms and Effects.—Cases of poisoning by foxglove are not very numerous. One was the subject of a criminal trial at the Old Bailey, in Oct. 1826. A quack was indicted for the manslaughter of a boy under the following circumstances: he prescribed for a trivial complaint six ounces of a strong decoction of the *leaves*. The boy was soon attacked with vomiting, purging, and severe pain in the abdomen. After some time, he became lethargic, and slept for several hours; in the night he was seized with convulsions. The pupils were dilated and insensible, the pulse was slow, small, and irregular; coma followed, and the boy died twenty-two hours after taking the poison. On *inspection*, the membranes of the brain were found much injected, and the mucous lining of the stomach was partially inflamed. The prisoner was acquitted of the charge, because he had only given his advice on the application of the friends of the deceased! (Ed. Med. and Surg. Jour. xxvii, 223.) For cases of recovery from a strong dose of the infusion, see Med. Gaz. xxxiv, 659; and L'Union Medicale, 24 Août, 1848. On the other hand, a case in which an infusion of the *root* proved fatal is reported in the Lancet, July 14, 1849, p. 31. Accidents sometimes occur from the medicinal use of the *tincture*. In a late number of the Medical Gazette is the account of a case in which from a dose of the tincture too frequently repeated, the person was attacked with restlessness, thirst, inflamed eyes, and other serious symptoms.

A young man having filled a quart pitcher with the leaves of foxglove, poured upon them as much boiling water as the pitcher would hold. Of this strong *infusion* he took a teacupful on going to bed, which caused him to sleep soundly. In the morning he took a second cupful (the infusion being much stronger), and he then went to his employment. He soon felt dizzy and heavy, began to stagger, lost his consciousness, and at length fell down in a state of syncope. On being conveyed home he vomited severely, and suffered extreme pain in the abdomen. When visited, he was conscious, complained of great pain in his head; the pupils were dilated, and the skin was cold, pallid, and covered with a copious perspiration. The pulse was low, about 40 in the minute,—three or four feeble pulsations being succeeded by a complete intermission of several seconds; and each stroke, though weak, was given with a peculiar "explosive shock." There was still great pain in the abdomen, with incessant and violent vomiting, no purging,—suppression of urine, and an abundant flow of saliva. Brandy and ammonia with warmth were employed, and after reaction had commenced, purgatives were administered. The man slowly recovered, but the pulse presented its peculiar beat and weakness for several days; and during this time the man could not bear the upright position.

In another instance, a young man swallowed a strong *decoction* of foxglove by mistake for purgative medicine. He was soon seized with vomiting, pain in the abdomen, and purging. In the afternoon he fell asleep. At midnight he awoke, was attacked with violent vomiting, colic, convulsions, dilated and insensible pupils; and his pulse was slow and irregular. He died twenty-two hours after taking the poison. (Wibmer, op. cit. Digitalis.) A few grains of the powdered leaves have been known to produce giddiness, languor, dimness of sight, and other nervous symptoms. A drachm has, however, been taken without causing death; but in this instance it produced the most violent vomiting. As indicative of the singular effect of this poison on the nerves of sensation, it may be stated that a coal fire appeared to the patient to have a blue color. A common effect of this poison is to produce great depression of the heart's action.

In the case of a man, æt. 50, the tincture, taken in medicinal doses for about twenty days, produced the following train of symptoms. (Med. Gaz. xxxi, 270.) The pulse, which, during a former use of the medicine, was reduced by ten or fifteen beats in a minute, sank almost to half its usual number. The patient was tormented by the most painful disquietude, so that, even in the night, he left the bed every moment, could not sleep, and with his eyes open conversed with persons who were not present. At the same time the pupils were dilated, the conjunctiva both of the eye and the lids was red; he had but little appetite, with great nausea, violent thirst, and dryness of the mouth; the alvine evacuations were scanty; the secretion of urine was increased. These phenomena, which obviously were merely the effects of the digitalis, had lasted six days, when the restlessness diminished, sleep returned, and the dilatation of the pupils disappeared. This case shows that digitalis possesses accumulative properties; and that it cannot be given for a long period medicinally without producing dangerous symptoms. Dr. Elliotson states that he has known persons who had been in the habit of taking this medicine for a long period, die very suddenly, as if from the accumulation of the poison in the system and its fatal action on the heart. The *appearances* met with after death are congestion of the brain and its membranes; inflammation of the mucous membrane of the stomach, and fluidity of the blood.

Fatal dose.—The medicinal dose of the infusion is from half an ounce to one ounce; of the tincture, from ten to forty minims; and of the powder, from half a grain to one grain and a half. The medicinal preparations vary considerably in strength, a fact which will explain why they have been administered in much larger doses than those here assigned, without producing dangerous effects. According to the late Dr. Pereira, twenty drops of the tincture were given to an infant, laboring under water on the brain, three times daily for a fortnight; without causing any untoward symptom; and he frequently prescribed for an adult, one drachm of the tincture three times daily for a fortnight without producing any marked effect. The tincture has been sometimes prescribed medicinally in doses of half an ounce to an ounce: and on one occasion two ounces were taken in two doses without giving rise to the slightest inconvenience. These facts show either that foxglove is not so powerful a poison as it is commonly supposed to be, or that the proportion of digitalia is liable to great variation in the alcoholic solution.

Treatment.—In a case of poisoning by foxglove, in addition to the free use of emetics, vegetable infusions containing tannic acid should be given. According to the researches of M. Homolle, this renders the active principle insoluble.

Analysis.—When foxglove has been taken in substance, it can be identified only by its botanical characters. In the form of infusion, decoction, or tincture, and when mixed with organic liquids, there are no chemical processes by which the nature of the poison can be determined.

DIGITALIA.—The active principle of foxglove is called *digitalia.* Its properties have been investigated by M. Homolle. (Journ. de Pharmacie, Janvier, 1845, 57.) The process for obtaining it is exceedingly complex. It is a white, inodorous, imperfectly crystalline substance. It is so intensely bitter, that it gives a sensible bitterness, to 200,000 parts of water; but the taste of digitalia itself is only slowly manifested, in consequence of its great insolubility. Cold water dissolves only 1-2000th, and hot water only 1-1000th part: it is much more soluble in alcohol and ether. When dissolved in either menstruum, it has neither an acid nor alkaline reaction. It does not form salts with acids. It immediately decomposes nitric acid, evolving nitrous acid vapors, and producing a rich orange-yellow colored solution, which acquires in a few days a golden-yellow tint. Sulphuric acid at first blackens it, but subsequently forms a brownish-black liquid, which passes in a few days to a red-brown, amethyst, and finally a rich crimson color. If a portion of the reddish-brown liquid be dropped into a small quantity of water, it gives to it a rich green color. Hydrochloric acid produces with it a yellow, speedily passing to a bright-green color. A dose of more than 1-16th of a grain could not be taken by an adult without causing symptoms of poisoning. This quantity was found to be equal in strength to about eight grains of the well-prepared powder of the leaves.

THORNAPPLE—(DATURA STRAMONIUM.)

Thornapple has decidedly the characters of a narcotico-irritant poison. All parts of the plant are poisonous; but the seeds and fruit are considered to be the most noxious. From a case published by Dr. Zechmeister, it would appear that even the *vapor* of the full-blown *flowers* may give rise to symptoms of poisoning. The case was that of a boy who breathed the vapor for some time in a close apartment. (Œsterreich Med. Woch. July 19, 1845.)

Symptoms.—The usual effects produced by this poison will be understood from the following cases. A woman, æt. 36, took two teacupfuls of infusion of stramonium, by mistake for senna tea. In about ten minutes she was seized with giddiness, dimness of sight, and fainting. In two hours she was quite insensible; the pupils were fixed and dilated, all the muscles of the body convulsed, the countenance flushed, and the pulse was full and slow. The stomach-pump was applied, and in the course of a few hours she recovered,—suffering, however, from indistinctness of vision and vertigo. (Med. Gaz. viii, 605.) The *seeds* of this plant have been known to produce furious delirium; and a case is mentioned by Sauvages, of an old man of sixty, who, after taking the poison, became intoxicated, maniacal, and lost the power of speech. He remained in a lethargic state for five hours. Several fatal cases are reported, one of which terminated in six hours. Dr. Thomson relates the case of a child, aged two years, who swallowed sixteen grains of the seeds. Maniacal delirium supervened; the symptoms resembled those of hydrophobia, and death took place in twenty-four hours. A case which occurred to Dr. Schlesier ended more fortunately. A boy, æt. 4, mistaking the fruit of the thornapple for the heads of poppies, ate a quantity of them. Dr. Schlesier saw him soon afterwards: his face was flushed, his eyes were glistening and in constant motion, the pupils much dilated, and the countenance was that of an intoxicated person. He sat up in bed quite unconscious, but continually babbling and occasionally starting up suddenly, his hands apparently directed at imaginary objects in the air. His pulse was very slow; there was no fever, but intense thirst and violent perspiration from incessant motion. Emetics and injections were administered, which had the effect of bringing away a large quantity of stramonium seeds; the boy fell into a sound sleep, and recovered on the following day. (Canstatt's Jahresb. 1844, v, 297.)

In the Lancet (April 26, 1845, p. 471), a case is quoted from the Boston Journal, in which three females swallowed an infusion of stramonium *leaves* for horehound. They were found lying in bed, stupid, unable to articulate, with a peculiar wildness of countenance, and flushed face: the pupils were dilated and insensible, the conjunctivæ highly injected, lips and tongue parched, no vomiting, breathing at times stertorous and labored, hands cold, with a trembling and slightly convulsive movement, great rigidity of the muscles of the neck and back, and occasionally active efforts at utterance. Stimulants were administered with benefit in two cases: the third proved fatal. Mr. Sobo met with the case of a child, æt. 5, who ate more than a drachm of the seeds slightly roasted. In about an hour he appeared much excited and delirious, pulse 120, face flushed, eyes of a dazzling lustre, and pupils dilated; there were convulsive motions of the limbs and neck, with thick frothy saliva about the mouth. Emetics were given, some stramonium seeds were ejected, and more were brought away in the evacuations by a full dose of castor oil. In three days the boy had perfectly recovered. (Med. Times, Oct. 9, 1847, p. 650. For other cases, see Prov. Journal, Dec. 24, 1851, p. 699; and Lancet, May 31, 1851, p. 599.) Paralysis and delirium have been witnessed among the symptoms, which on the whole bear a strong resemblance to those occasioned by belladonna. The detection of the seeds in the vomited matters or in the fæces, will be a certain means of distinction.

Among the most recent cases of poisoning by the seeds is the following: A boy, æt. 5, ate some stramonium seeds, with a portion of the plant. Soon afterwards, it was observed that his face was flushed, and that he staggered as if intoxicated. He vomited and threw up about thirty seeds. His skin was hot and red, the countenance had a wild and staring expression, the pupils were nearly fully dilated, and insensible to light. The child was restless, in a state of raging delirium, and biting with fury at those who attempted to restrain him. He was unable to stand, and in a state resembling St. Vitus's dance. The pulse could not be counted. The breathing was hurried and gasping. He was incessantly talking, but without articulating distinctly, and he appeared to be driving away from him imaginary objects. Emetics produced the vomiting of more seeds, and in an hour he began to articulate. He slept restlessly for two hours. Some seeds were passed in evacuations from the bowels. In four hours the symptoms had abated, and the boy gradually improved. The pupil did not recover its natural state until after three days. (New York Journal of Medicine, 1856; and Brit. and For. Med. Rev. 1857, vol. xix, p. 497.)

In a case which became the subject of a trial at Osnabrück, a woman administered to her mother a decoction of the bruised *seeds* of the thorn-apple, of which it was supposed there were about 125. She very soon became delirious, threw her arms about, and spoke incoherently: she died in seven hours. (Henke, Zeitschrift der S. A. 1837, i, H.) The seeds retain their properties notwithstanding their exposure to heat: thus the smoking of stramonium seeds is attended with danger. In the return of the Registrar-General for April, 1856, there is the record of one death from this cause. One of the methods of poisoning adopted by the Hindoos, not so much with the intention of destroying life as of facilitating the perpetration of robbery, consists in administering to persons either the powdered seeds, or a strong decoction of them, in curry, or some other highly flavored article of food. Delirium and insensibility soon follow, and sometimes death is the result; but no suspicion of the real cause appears to be excited.

Dr. Chevers has given a very complete account of the Hindoo system of poisoning by *dhatoora.* (Med. Jur. for India, 1856, pp. 121, 549, 591.) It appears that the Datura fastuosa and alba are the principal sources of the poison in India. The Thugs have employed this poison with the object of rendering their intended victims helpless. As it is administered by skilled professional poisoners in India, it causes a profound lethargy resembling coma, with dilated pupils. The symptoms may continue for two days, and yet recovery take place. It is remarkable that the cases rarely prove fatal. Out of fifty-one instances of poisoning by dhatoora, at the Bombay hospital, recorded by Dr. Giraud, one proved fatal, and four only presented very alarming symptoms. Dr. Chevers notices the early occurrence of insensibility. A man drank two mouthfuls of a poisoned liquid, complained of a bitter taste, and fell down insensible within forty yards of the spot where he had drunk the liquid, and did not recover his senses until the third day after. (Op. cit. p. 137.) In these cases, probably the poison is given in a large dose, either in solution or in very fine powder. The first stage of poisoning is commonly marked by delirium, the patient is restless and wanders about as if in search of something, but from giddiness or great muscular weakness, he is unable to walk or even to stand; he talks incoherently, laughs wildly, moves about as if to avoid spectra, and picks or catches incessantly at real or imaginary objects. His antics are sometimes of the most varied and ludicrous kind. The pupils are invariably dilated, and the spectra are illusions depending on disordered vision. In the second state of poisoning, there is either great drowsiness or complete stupor, sometimes passing into utter insensibility, with stertorous breathing. The third stage of final delirium is similar to the first. (Op. cit. p. 593.) An extract of datura is probably used as one of the methods of "hocussing" persons by thieves in this country. The dilatation of the pupil, with the peculiar train of symptoms, would distinguish this state from ordinary intoxication. The bitter taste of the liquid might excite suspicion; but if the person is already partially intoxicated, he may be incapable of making any observation of this kind.

The *local application* of the bruised leaves, seeds, or fruit to an abraded portion of skin, may give rise to all the effects of poisoning.

Death may take place, even although the whole of the seeds have been ejected, provided they have remained in the body for a sufficiently long period to allow of the absorption of the poisonous principle. This happened in a case reported by Mr. Duffin, that of his own child, æt. 2, who swallowed about one hundred seeds of stramonium, weighing sixteen grains: the usual symptoms were manifested in an hour, and the child died in twenty-four hours, although twenty seeds had been ejected by vomiting, and eighty by purging. (Med. Gaz. vol. xv, 194.)

The *extract* of stramonium possesses the properties of the seeds, producing, in an over-dose, dryness of the throat, intoxication, and delirium. Dr. Traill has known two cases of poisoning by this substance, in one of which eighteen grains of the extract were taken by mistake for extract of sarsaparilla. (Outlines, 141.) The medicinal dose of the extract is about a quarter of a grain; of the powdered seeds half a grain; and of the leaves, one grain.

Appearances.—In a well-marked case of poisoning by stramonium seeds, in which death took place in less than eight hours, Mr. Allan found the following appearances: great congestion of the vessels of the brain and its membranes, the brain firm and highly injected, choroid plexus turgid, ventricles containing serum, substance of the lungs congested, the heart flaccid. The stomach contained about four ounces of digested food mixed with eighty-nine seeds of stramonium. There were two patches of extravasation in the mucous coat—one on the larger curvature, and the other near the pylorus. Many seeds and fragments were also found in the intestines. (Lancet, Sept. 18, 1847, p. 298.) In the Osnabrück case there were marks of diffused inflammation about the cardia. In Mr. Duffin's case (supra) there was nothing remarkable in the condition of the brain or its membranes: no seeds were found in the intestinal canal.

Analysis.—The *seeds* of stramonium, from which accidents have most frequently occurred, are flattened, kidney-shaped, rough, and of a dark-brown or black color.

DATURIA.—The poisonous properties of this plant are owing to the presence of the alkaloid *daturia*, which forms about one per cent. of the dried vegetable. This substance crystallizes in colorless quadrangular prisms or needles: it has a bitter taste, becoming acrid, and resembling that of tobacco. It has the same elementary composition as atropia. (Schwarzkopf and Planta.) It is highly poisonous: the eighth of a grain killed a sparrow in three hours. When dissolved and placed on the eye of an animal, it causes excessive dilatation of the pupil, which lasts for some days. It is readily dissolved by boiling water, and the solution has a strong alkaline reaction. In its chemical properties it closely resembles hyoscyamia and atropia.

The absorption of this poisonous alkaloid is doubtless the cause of the symptoms. Mr. Allan, in the case above related, states that he obtained from six ounces of urine, taken from the bladder of the deceased, crystals of daturia; but they appear to have been of an entirely different form, *i. e.* pentahedral or polyhedral plates, instead of quadrangular prisms. They resembled daturia only in causing dilatation of the pupil, when dissolved in water and the solution was dropped into the eye. Their form appears to have been that assigned to cystin by microscopical observers. (Bird's Urinary Deposits, 146.)

LABURNUM—(CYTISUS LABURNUM.)

Symptoms and Effects.—The bark and seeds of the common LABURNUM contain an active poison called *Cytisine.* A case of poisoning by the *bark*, which was the subject of a trial at Inverness, has been reported by Dr. Christison. (Ed. Med. and S. J. Oct. 1848.) A youth, with the intention of merely producing vomiting in one of his fellow-servants, a female, put some dry laburnum bark into the broth which was being prepared for their dinner. The cook, who remarked a "strong peculiar taste" in the broth, soon became very ill, and in five minutes was attacked with violent vomiting. The account of the symptoms is imperfect; for the cause of them was not even suspected until six months afterwards. The vomiting continued thirty-six hours; was accompanied by shivering,—pain in the abdomen, especially in the stomach,—and great feebleness, with severe purging. These symptoms continued, more or less, for a period of eight months; and the woman fell off in flesh and strength. At this period she was seen by a physician, who had been called on by the law-authorities to investigate the case. She was then suffering from gastro-intestinal irritation, vomiting after food, pain in the abdomen, increased by pressure, purging, tenesmus, and bloody evacuations, with other serious symptoms. The medical opinion was that she was then in a highly dangerous state. The woman did not eventually recover until the following April. There was no doubt, from the investigation made by Dr. Ross and Dr. Christison, that her protracted illness was really due to the effects of the laburnum bark.

Some experiments were then made on the action of the poison on animals. A teaspoonful of the powder of dry laburnum bark was administered to a cat. Soon afterwards it writhed, apparently in great pain; in a short time it vomited violently, and, although languid and dejected for the rest of the day, it quickly recovered. Sixty-nine grains of the same powder were given to a dog. In ten minutes it whined and moaned, vomited violently, and soon got well. On a second occasion, twenty grains were found to act as a powerful emetic upon the animal. An ounce of the infusion of laburnum-bark, containing the active matter of sixty-two grains, was introduced by a catheter into the stomach of a full-grown rabbit. In two minutes the animal looked quickly from one side to the other, twitched back its head twice or thrice, and instantly fell upon its side in violent tetanic convulsions, with alternating emprosthotonos and opisthotonos, so energetic, that its body bounded with great force upon the side, up and down the room. Suddenly, in half a minute more, all movement ceased, respiration was at an end, the whole of the muscles became quite flaccid, no sign of sensation could be elicited, and the animal died within *two minutes and a half* after the poison was injected into the stomach. The body was opened in two minutes more, and the heart was found gorged with blood, but contracting with some force. The stomach was filled with green pulp, soaked with the infusion. No morbid appearance was visible anywhere. In repeating this experiment, one rabbit died in half an hour, another in three-quarters of an hour, after small doses of the infusion were injected into the stomach; and a third rabbit speedily died after eating greens merely impregnated with the infusion. (Ed. Med. and Surg. Journal, 1843, vol. lx, p. 303.) In all these instances convulsions were the leading symptoms produced. The same effects are popularly ascribed to the leaves, young pods, and seeds of the tree; but no experiments have been performed with them. The facts here detailed show that laburnum-bark is an energetic poison.

In reference to poisoning by the *seeds* there are but few instances recorded. Dr. Traill has described two cases, and Mr. Rake, a former pupil, has communicated to me a case of poisoning by the pods and seeds of laburnum which occurred in September, 1851. Two children, the one aged two, and the other three years, had been seen playing together, and on returning home they appeared unwell, and soon afterwards vomited. They had been seen with laburnum pods in their hands, and some seeds with portions of the pods were mixed with the vomited matter. Both children were pale and exhausted, with a slow and somewhat feeble pulse. The pupils were natural. An emetic was given, but no more seeds were ejected: the pulse increased in volume and frequency, and the next day the children had recovered their usual health. In October, 1856, twelve children, at Otley, in Yorkshire, were attacked with rigidity of the limbs and other symptoms of poisoning in consequence of having swallowed these seeds. They recovered under the use of emetics. (Lancet, Nov. 1, 1856, p. 497.) Mr. Barber, of Stamford, communicated to me, in June, 1848, the particulars of a case which shows that even the *flowers* of this plant are highly noxious. A child between three and four years of age ate twelve laburnum flowers, and in about fifteen minutes it complained of sickness and severe pain in the stomach. The child vomited a quantity of mucus mixed with the yellow petals of the laburnum. An emetic was given: this cleared the stomach, and the child recovered. There was no purging. (Guy's Hosp. Reports, Oct. 1850, p. 219.) A case in which a child suffered from symptoms of a nervous kind by reason of its having eaten laburnum flowers, is described by Mr. North in the Medical and Physical Journal, vol. lxii, page 86.

Analysis.—The bark, flowers and seeds could not be identified only by their botanical characters. When administered in powder, infusion, or decoction, there are no chemical processes known by which the poison may be detected. A decoction of the bark forms a clear light brown liquid having an acid reaction. It strikes a dark olive-green color with a persalt of iron. Nitric acid renders it lighter. Acetate of lead precipitates it, but the precipitate has none of the properties of meconate of lead.

YEW—(TAXUS BACCATA.)

Symptoms and Effects.—It has been long known, that the berries and leaves of the yew-tree are poisonous to cattle; they act very energetically, and produce death in a few hours, sometimes without vomiting or purging. It is stated by Dr. Percival, that a tablespoonful of the *fresh leaves* was administered to three children of five, four, and three years of age as a vermifuge. Yawning and listlessness soon suceeeded; the eldest vomited a little, and complained of pain in the abdomen, but the other two suffered no pain. They all died within a few hours of each other. A case of poisoning by the *berries* of this tree, was published a few years since by Mr. Hurt, of Mansfield. A child aged three years and a half, ate a quantity of yew berries about eleven o'clock. In an hour afterwards, the child appeared ill, but did not complain of any pain. It vomited part of its dinner, mixed with some of the berries. A medical man was sent for, but the child died in convulsions before he arrived. On inspection, the stomach was found filled with mucus, and the half-digested pulp of the berries and seeds. There were patches of redness in the mucous membrane, and this was so much softened that it could be detached with the slightest friction. The small intestines were also inflamed.

In March, 1845, a case was reported to the Dublin Pathological Society by Dr. Mollan, in which a lunatic had died from the effects produced by *yew-leaves*. The deceased was observed chewing the plant, probably from that perversion of appetite so commonly observed in insanity, and before the attendants had taken it from him, he had succeeded in swallowing a portion of the masticated juice. He was soon afterwards seized with giddiness, sudden prostration of strength, vomiting, coldness of the surface, spasms, and irregular action of the heart. He died in fourteen hours. On inspection, the stomach was found much distended—it contained some yew-leaves. There was emphysema in the submucous tissue, but no other abnormal change; there was some thickening with opacity of the arachnoid, which might have been due to the insanity. (Dub. Hosp. Gaz. May 15, 1845, p. 109.)

There is no doubt that the yew is a cerebro-spinal poison. The symptoms produced by the *leaves* and berries are pretty uniform in character: convulsions, insensibility, coma, dilated pupils, pale countenance, small pulse, and cold extremities are the most prominent. Vomiting and purging are also observed among the symptoms. In two cases, the subject of one—a girl, about five years of age—died in a comatose state in four hours after she had eaten the *berries*, and the other, a boy, æt. 4 years, died nineteen days after taking the berries, obviously from severe inflammation of the bowels. The immediate symptoms in the boy were vomiting, purging, coma, convulsions, dilated pupils, hurried respiration, a small pulse, and a cold skin. (See Prov. Journal, Nov. 29, 1848, p. 662, and Dec. 27, p. 708.)

There is a vulgar but erroneous notion that the yew-leaves are not poisonous when fresh, and that in any case they act only mechanically. A case related above shows the fallacy of the opinion, and the other cases prove that there is a specific poison in the yew, since it exists in the berries as well as in the leaves. If cattle recover from the primary effects on the nervous system, they are liable to die, after several days, from inflammation of the bowels. On one occasion I examined the viscera of an ox, which had obviously died from the effects of yew-leaves. In some parts of the intestine gangrene had taken place.

The nature of the poisonous principle is unknown, and it is not certain whether, with respect to the berry, the poison is lodged in the pulp or the seed, although it is most probably in the latter. *Infusion* of yew-leaves, which is properly called yew-tree tea, is sometimes used for the purpose of procuring abortion by ignorant midwives. A case of death from a person drinking this infusion, is reported in the registration returns for 1838-9. In the returns for 1840, there is also one death of a female, æt. 34, referred to her having eaten the berries of the yew. The subject of poisoning by yew-leaves, in reference to their employment for purposes of abortion, has been lately investigated by MM. Chevalier, Duchesne, and Reynal. (See Ann. d'Hyg. 1855, vol. ii, pp. 94 and 335.)

Analysis.—The leaves or berries may be found in the stomach. The yew and the savin are the only coniferous poisons which grow in this country. The apex of the leaf of the yew is lancet-shaped, that of the savin acuminated; the yew-leaf does not possess the powerful odor of savin when rubbed. Yew-berries are seen in autumn; they are about the size of a pea, of a light-red color, dull on the surface, and translucent. They are open at the top, allowing a hard brown kernel to be seen. This is of an ovoid shape, and it forms the greater part of the berry. The fine red skin contains a colorless or remarkably viscid or adhesive juice, which reddens litmus paper, and has a nauseous sweetish taste.

PRIVET—(LIGUSTRUM VULGARE.)

The Privet is not commonly enumerated among vegetable poisons. No reference is made to this plant in the works of Wibmer, Orfila, Christison, and other writers on toxicology, and yet it would appear from the subjoined cases, for the brief particulars of which I am indebted to Mr. Ward, of Ollerton, that the *berries* may exert a poisonous action. In December, 1853, three children ate the berries of the privet; two of them, a boy of three years of age and a girl of six, eating them rather freely. They suffered from violent purging, and when seen by a medical man, the little boy was found pulseless, cold, and before death was frequently and violently convulsed. The girl was in a state of collapse, but rallied a little under treatment: she soon afterwards died convulsed. The surviving child, who had only tasted the berries, did not suffer, and she was enabled to point out the shrub, the berries of which they had gathered. So far as I know, these are the only cases on record in which the berries of the privet have proved fatal. According to Loudon, they are eaten by birds when other sources of food fail.

APPENDIX.

Page 360. *Reinsch's process for arsenic.*—Dr. Guy, of King's College, has proposed a new method for subliming the arsenical deposit obtained by this process. He collects the sublimate on microscopic glass, so that it may be at the same time examined by the microscope and treated by the usual chemical tests required for corroboration. The reader is referred for the details to Beale's Archives of Medicine, No. 3, 1858.

Page 388. *Arsenical paper-hangings.*—The mode in which the arsenical pigment may affect persons who inhabit rooms has been recently determined by experiment. In December, 1858, I examined the fine *dust* on some books in a book-case secured with glass doors, and found therein a well-marked quantity of arsenic. The room had been papered with an arsenical green paper, which it was supposed had caused some symptoms resembling arsenical poisoning in a friend who occupied it. The books had not been dusted for a period of three years. From a shop in the city, the walls of which were covered with this paper, I procured some dust which yielded a large proportion of arsenic. In the house of a friend, in which an arsenical paper was hung, I found arsenic in the dust on the cornices and on the gold moulding of the picture-frames. (See Med. Times and Gazette, Jan. 1st, 1859, p. 5.) Care was taken in these experiments to remove the dust without touching the wall-papers. In most of these cases a large proportion of the surface of the paper was *unglazed:* and the pigment was roughly laid on. It is, therefore, obvious that the atmosphere of rooms papered with arsenical paper-hangings must be more or less contaminated with the fine particles of the green pigment removed from the wall by mechanical causes. Changes of a hygrometric and thermometric nature may affect this porous pigment, and render it more easily detached by currents of air, vibration, &c. The poisonous particles may thus be received into the lungs; and although the quantity breathed at any one time may be small, it is certainly not advisable that, merely for the sake of a green color, persons should be exposed to breathe day by day arsenic in any proportion. The deaths of the members of the Arzone family (ante, p. 119) appear to receive an explanation from the results of these analyses.

INDEX.

[*The principal subjects are in small capitals, the cases in italics.*]

Abercromby, Miss, case of, 135.
Absorbed arsenic, diffusion of, 55; elimination of, 35, 50, 370.
Absorption, of poisons, 35, 38; of arsenic, 45; of antimony, 56; of silver, 65; of lead, 65; of mercury, 67; prussic acid, 71; of the alkaloids, 72; of morphia, 73, 560; of strychnia, 74; of alcohol, 79; conclusions respecting, 90; loss of poisons by, 176; of sulphuric acid, 237; of nitric acid, 263; of oxalic acid, 293; of potash, 299; of lead, 444; of copper, 470.
Absorption of lead by vegetables, 456.
Acacia leaves, prussic acid in, 624.
Acrid poisons (see Irritant poisons), 104.
Accumulative poisons, 152.
Acetate of lead, 428; of copper, 461; of morphia, 546; of strychnia, 691.
Aceto-arsenite of copper, 329, 385, 743.
ACID, sulphuric, 223; nitric, 252; muriatic or hydrochloric, 265; phosphoric and phosphorous, 71, 274; oxalic, 276; tartaric, 294; arsenious, 319; arsenic, 391; meconic, 551; prussic, 562; sulpho- and ferrocyanic, 632.
Acids, mineral, 223; vegetable, 276.
Aconitina, 724.
Aconite (see Monkshood), 718.
Aconitum napellus, 718.
Acute poisoning, 151.
Administration, medical evidence of, 188.
Adnam v. *Betts*, case of, 450.
Æthusa Cynapium, 716.
Agaricus campestris, 665; muscarius, 667.
Albuminuria and mercurial poisoning, 405.
ALCOHOL, absorption of, 79; poisoning by, symptoms, 635; effects of the vapor, 636; chronic poisoning by, 637; appearances after death, 638; fatal dose, 640; period of death, treatment, 640; analysis, 641; in the tissues, 642.
Alcoholic liquids, poisoning of, 643.
Alexander, Dr., case of, 49, 344.
Algaroth powder, 485.
Alkaline poisons, 295.
Alkaline salts, poisoning by, 303.
Alkaloidal poisons found in the blood, 41.
Almonds, bitter, poisoning by, 605; sweet, prussic acid in leaves of, 624.
Almond flavor, 606, 615.
Alum, action of, 310.
Amanita muscaria, 667.
Amorphous phosphorus, 318.
Ammonia, poisoning by, in liquid and vapor, 299; carbonate of, 302.
Ammonio-chloride of mercury, 422.
Amygdaline, prussic acid produced from, in the blood, 87; in organic mixtures, 600.
Amylene, noxious effects of, 654.
ANALYSIS, CHEMICAL, rules for conducting, 172; objects of a, 182; delicacy of, 189; fallacies in, 192, 559; value of evidence from, 210.
Angustura, false, bark of, 677.
Animals, evidence from experiments on, 195; poisoned flesh of, 200.
Animal charcoal as an antidote in poisoning, 708.
Animal food, poisonous effects of, 200.
Animal irritants, 499.
ANTIMONY, absorption of, 56; deposition of, in the tissues, 57; elimination of, 59; inferences from its discovery in the body, 63; tartarized, poisoning by, 476; symptoms caused by, 476; chronic poisoning, 479; external application, 479; appearances after death, 479; fatal dose, period of death, 481; chemical analysis, 483; in organic liquids, 484; in the tissues, 485; arsenic in the compounds of, 488; chloride or butter of, 489.
Antimonial and arsenical deposits, distinction of, 354.
Antimonial compounds, arsenic in, 488.

Antimonial wine, 476.
Antiarin, 678.
Antiaris toxicaria, 678.
Apoplexy, symptoms of, 130; a cause of death in infants, 132; from excessive drinking, 636.
Apparatus, poisons in, 190.
Appearances after death, evidence from, 153.
Appendix cœci, death from foreign bodies in, 164.
Apple pips, alleged presence of prussic acid in, 624.
Aqua fortis (see Nitric acid), 252.
Aqua lauro-cerasi, 624.
Aqua Toffana, 177.
Argol, 305.
Arseniate of soda, 392.
Arsenic acid, 391.
Arsenic, metallic, 390.
ARSENIC, detected in the blood, organs, and secretions, 45; deposited in the liver, 46; period required for the absorption and elimination of, 47; not affected by habit, 96; nervous symptoms caused by, 105; chemical change of, in the dead body, 181; poisoning by, 319; taste of, 320; not a corrosive poison, 321; solubility of, 322; symptoms caused by, in acute poisoning, 322; variable nature of the, 105, 326; chronic poisoning by, 328; not an accumulative poison, 331; effects of external application, 333; appearances after death from, 335; period required for inflammation and ulceration, 337; absence of inflammation, 338; quantity required to destroy life, fatal dose, 339; recovery from large doses, 342; period of death, 342; influence of quantity, 344; treatment, 344; chemical analysis, 347; reduction process, 348; objections, 349; tests for, in solution, 349; objections, 350; gaseous test for, 351; Marsh's process, or hydrogen test, 353; deposits of, known from antimony, 355; in zinc and sulphuric acid, 355; Reinsch's process, 358; detection of, in organic liquids, 360; detection of, in the tissues, 363, 366; not detected, 369; quantity found, 187; diffusion of, in the body, 55; normal, 371; in articles of food, 372; detected in the dead after long periods, 374; in the soil of cemeteries, 376; preservative effects of, 380; its presence in grain, 373; quantitative analysis, 368; sulphurets of, 393; chloride of, 395; in lead shot, 427; in antimonial preparations, 488.
Arsenic, alleged cosmetic use of, 98.
Arsenic, eating, alleged practice of, 94.
Arsenical green, poisoning by, 385.
Arsenical vapors, 119; alleged death from, 216; effects of, 382; paper hangings, 388, 743.
Arsenite of potash, poisoning by, 383; of soda, 341, 385; of copper, 329, 385, 743.
Arsenuretted hydrogen, 353, 395.
Arzone family, case of the, 119.
Ascarides, 165.
Asiatic cholera mistaken for poisoning, 123.
Asphyxia, syncopal, 145; produced by sulphuric acid, 228.
Atelectasis mistaken for narcotic poisoning, 133.
Atlee family, cases of the, 52.
Atropa belladonna, 725.
Atropia, 730.

Bacon, case of, 157, 367, 381, 420.
Bainbridge, Dr., case of, 585.
Baked meats poisoned by lead, 446.
Barium, chloride of, poisoning by, 205.
Battley's Sedative Solution, 542.
Bean of St. Ignatius, 677.
Beard, case of, 179.
Bear's-foot, poisoning by, 508.
Belaney, case of, 588.
BELLADONNA, poisoning by the berries of, symptoms, 725; by the root and leaves, 727; extract, 729; local action, 729; appearances after death, 730; analysis, 731.
Bentinck, Lord George, case of, 145.
Bichloride of Mercury (see CORROSIVE SUBLIMATE), 398.
Bichromate of potash, 497.
Bicyanide of mercury, 424.
Binarseniate of potash, 393.
Binoxalate of potash, 304.
Bismuth, poisoning by, 497.
Bitartrate of potash, 305.
BITTER ALMONDS, noxious effects of, 605; essential oil of, 605; quantity of prussic acid obtained from, 616.
Bitter almond water, poisoning by, 619.
Bitter-sweet, 670.
Black arsenic, 320.
Black drop, 541.
Black hellebore, 508.
Blandy, Miss, case of, 152.,
Blistering flies (see CANTHARIDES), 511.
Blistering plaster, poisoning by, 512.
Blood, poisons detected in the, 38; condition of, in poisoning, 86; action of poisons, when injected into the, 38; production of poisons in, 87.
Blue arsenic, 320.
Blue-rocket, poisoning by, 718.
Blue-stone, 459.
Blue vitriol, 21, 459.
Bocarmé Count, case of, 187, 661.
Bodle, case of, 173.

Boiling liquids, effects of, when swallowed 34.
Boletus Cervinus, alleged death from, 207.
Bond, Mr., case of, 115, 140, 683.
Boulet, case of, 233.
Boughton Sir T., case of (see *Donellan*), 115, 191, 622.
Body, rules for inspecting, 167.
Boyden, Reg. v., 732.
Boys, case of, 50.
Brain and spinal marrow, diseases of, mistaken for narcotic poisoning, 144.
Brandy, poisoning by, 636.
Bread, arsenic in, 342; poisoned by copper, 474; by lead, 430; by antimony, 486.
Brennan, case of, 237.
Brettle, case of, 117.
Brucia, properties of, 705.
Brunswick green, 462.
Brussels lace whitened by lead, 433.
Burdock, case of, 353.
Burnett's liquid, poisoning by, 492.
Burton, case of, 370.
Burroughs, Reg. v., 565.
Buswell, Judith, case of, 569.
Butter of antimony, 489.
Butterfield, case of, 152, 216, 403.

Cabalonga, 677.
Cadaveric imbibition of poisons, 68.
Calabar, or ordeal bean, 678.
Calamine, 495.
Calmia latifolia, 202.
Calomel, fatal salivation from, 402; noxious effects of, 421.
Camphor, poisoning by, 654; analysis, 656; compound tincture of, 540.
Cancer of the mouth, 405.
Cancrum oris, 405.
CANTHARIDES, poisoning by, 511; symptoms, 511; chronic poisoning by, 513; appearances after death from, 514; analysis, 515.
Cantharidine, 515.
Capsules, leaden, poisoning by, 451.
Carbonates, alkaline, poisoning by, 296; of lead, 431; of copper, 463; of zinc, 495.
Carbonic oxide, death from, mistaken for poisoning by arsenic, 216.
Cassava, bitter, prussic acid in, 626.
Castaign, Dr., case of, 547.
Cattle poisoned by lead, 455.
Caustic alkalies, 296.
Caustic, lunar, 495.
Cellular tissue, action of poisons through the, 36.
Cemeteries, presence of arsenic in the soil of, 376; copper in, 472.
Cerebral poisons, 103, 153, 518.
Cerebro-spinal poisons, 706.
Ceruse, poisoning by, 431.
Chambers, Dr., case of, 585.
Charcoal powder in poisoning, 708.
Chemical analysis, evidence from, 169; convictions without, 169; failure of, 553; mistakes in, 559.
Cherry kernels, prussic acid yielded by, 624.
Cherry-ratafia, 624.
Cherry-laurel water, 621.
Chesham, case of, 339, 367, 375.
Chloride of arsenic, 395; of mercury, 398; of antimony, 489; of zinc, 492; of tin, 495; of gold, 495.
Chloroform liquid, effects of, 651.
CHLOROFORM vapor, symptoms, 648; appearances, 649; fatal dose, period of death, 652; treatment, analysis, 653; in the blood and tissues, 653.
Cholera mistaken for poisoning, 124.
Chromate of lead, 432.
Chromium, poisoning by, 497.
Chrome yellow, 432.
Christmas rose, 508.
Chronic poisoning, 151; by sulphuric acid, 235; by arsenic, 328; by mercury, 402; by lead, 433; by copper, 462.
Cinnabar, 424.
Cicuta virosa, 711.
Cicuta maculata, 713.
Cider, poisoned by lead, 447.
Cleansing liquids poisonous, 341.
Clothing, acid stains on, 247.
Cluderay, case of, 27.
Clysters, poisonous, 111, 526.
Cocculus indicus, 27, 662.
Cochlearia armoracia, characters of the root, 724.
Codeia, poisoning by, 548, 552.
Coins, swallowing of, 29.
Colchicum autumnale, poisoning by, 505.
Colchicina, 507.
Cold water, effects of, 25.
Colic mistaken for lead poisoning, 129.
Collica pictonum, 433.
Colic, painters', 433; from copper, 464.
Common salt action of, 18.
Compound poisoning, 215.
Concussion of the brain, known from alcoholic poisoning, 638.
Confectionery poisoned, 386.
Conium maculatum, 805.
Conia, 710.
Connell, Reg. v., 237.
Convulsions, tetanic, mistaken for poisoning, 136; causes of, 142; a frequent cause of death in children, 143.
Cook, J. P., case of, 63, 114, 137, 185, 697.
Copper coin, death from, 29.
COPPER, poisoning by, 458; symptoms, 459; external application, 464; ap-

pearances after death, 465; fatal dose treatment, 466; sulphate of, 22, 100, 459; chronic poisoning by, 462; arsenite of, 385; analysis of the salts of, 467; in organic liquids, 468; in the tissues, 470; in articles of food, 473; action of water on, 473; normal, 471; in the soil of cemeteries, 472.
Copperas, white, 491; green, 23, 491.
Copper, flesh poisoned by, 201.
Copper vessels, food poisoned by, 462.
Coriaria myrtifolia, 202.
Corrosive poisons, 104.
CORROSIVE SUBLIMATE, poisoning by, 398; taste and solubility, 398; symptoms of poisoning by, 398; chronic poisoning by, 402; its effects compared with arsenic, 409; salivation from, 403; external application of, 407; appearances after death from, 409; fatal dose of, 411; period at which death takes place, 412; treatment, 413; chemical analysis, 414; in organic liquids, 418; in the tissues, 419; not detected, 420; quantitative analysis, 421.
Cosmetics, poisonous, 437.
Cosmetic use of arsenic, 98, 335.
Cow's milk poisoned by lead, 66.
Cream of tartar, 305.
Cronin, case of, 620.
Croton seeds and oil, 503.
Crown Prince of Sweden, case of the, 112.
Curara poison, 679.
Curarina, 680.
Custaria bark, nux vomica mistaken for, 677.
CYANIDE OF POTASSIUM, vapor of, 564; poisoning by, 626; symptoms, 627; appearances after death, 629; local action, 630; fatal dose and period of death, 631; chemical analysis, 631.
Cyanide of mercury, 424, 634; of iron, 634; of silver, 634.
Cytisus Laburnum, 739.
Cytisine, 739.

Dalby's carminative, 539.
Daniel, case of, 423.
Darnel seeds, poisonous effects of, 664.
Datura stramonium, symptoms and appearances caused by, 735; analysis, 738.
Daturia, 738.
Dazley, case of, 339.
Deadly poison, meaning of, 21.
Deadly nightshade, 725. See BELLADONNA.
Dead-tongue (see Œnanthe Crocata), 713.
Deaths, sudden, causes of, 150.
Delirium tremens a result of poisoning by alcohol, 637.
Deposition of poisons in the tissues, 43, 52.
Deposits of arsenic and antimony, 354.
Destructive thing, legal meaning of, 28.
Diacetate of lead, 430; of copper, 461.
Dickman, Reg. v., 277.
Diffusion of poisons in the living body, 55; in the dead body, 68.
Digitalis purpurea (see Foxglove) 732.
Digitalia, 734.
Disease, influence of, on the action of poisons, 98; mistaken for poisoning, 109, 112; action of opium increased by, 109; of the heart mistaken for poisoning, 145; of the brain and spinal marrow, 144; resembling irritant poisoning, 123; neurotic poisoning, 129.
Distension of the stomach, death from, 146.
Distilled water, action of, on lead, 451; poisoned by lead, 442.
Donnall, case of, 351.
Dore and *Spry*, case of, 210.
Dove, case of, 197, 686, 703.
Dover's powder, 541.
Donellan, case of, 622.
Dripping poisoned by lead, 446.
Dulcamara, 670.
Dyes, hair-, containing lead, 437.
Dyer's spirit, 495.

Echidnine, action of, through a wound and the stomach, 36.
Edney, case of, 205.
Edwards, case of, 438.
Elimination of poisons, 44; loss of poisons by, 176, 237.
Elixir of vitriol, 249; paregoric, 540.
Embden groats, poisoned by lead, 450.
Emerald green, poisoning by, 329, 538, 743.
Emetic tartar, 476.
Enemata, poisoning by, 111, 526.
English cholera mistaken for poisoning by arsenic, 124.
Enteritis, symptoms of, 125.
Entozoa, poisons detected in, 55.
Epilepsy, symptoms of, 133.
Epileptics, Parisian, cases of, 566.
Epsom salts, fatal effects of, 19.
Ergot of rye, 44, 665.
Essential oil of bitter almonds, 605.
Ether, liquid, symptoms and effects, 647; fatal dose, treatment, 647; analysis, 648.
ETHER VAPOR, effects of, 643; appearances in death from, 645; absorption, chemical analysis, 648.
Evidence of poisoning, from symptoms, 106; from discovery of poison in the food, 122; from appearances after death, 153; from chemical analysis, 169; from experiments on animals, 202.
Exosmosis of poisons, 69.
Experiments on animals, value of evidence from, 199, 202.

Extract, Goulard's, poisoning by, 430.
Extract of poppies, 539.

Fallacies in chemical analysis, 191, 455, 559.
False angustura bark, 677.
Fasting, effects of, resemble poisoning, 118.
Fat poisoned by lead-glaze, 446.
Feigned poisoning, 122.
Ferrocyanic acid, action of, 632.
Ferrocyanide of potassium, 43, 632.
Filings, iron, irritant action of, 29.
Fine-leaved water-hemlock, 716.
Fish poisoned by copper, 462; by cocculus indicus, 663.
Fisher, Reg. v. 606.
Fleming's tincture of aconite, 721.
Flesh of poisoned animals poisonous, 201.
Flour, poisonous, 373, 448.
Fly-paper, 393.
Fly-powder; 390; water, 391.
Fly-mushroom, 667.
Fœtid hellebore, 509.
Fœtus, poisons detected in the, 55.
Food, poison detected in, 122; poisoned by copper, 120, 201; poisonous, 202; lead in, 446.
Fool's parsley, 716.
Forester, *Major*, case of, 526, 560.
Forty, case of, 721.
Foster, case of, 206.
Fougnies, case of, 187, 661.
Fowler's mineral solution, 383.
Foxglove, purple, poisoning by, 732; symptoms, analysis, 734.
Freeman, case of, 568.
Frog-test for strychnia, 197.
Fruits, preserved, poisoned by lead, 450; by copper, 473.
Fungi, poisoning by, 665.
Fungin, 665.

Gall-bladder, rupture of, death from, mistaken for poisoning, 147.
Game, poisoned, 200.
Gangrene of the mouth, 405.
Garden nightshade, 670.
Gardiner, *Dr.*, case of, 686.
Garland, case of, 382.
Gases, poisonous action of, 106.
Gastritis, symptoms of, 125.
Gastro-enteritis, 125.
Gaylor, Reg. v. 309.
Geerings, cases of the, 49.
Gelatinized perforation of the stomach, 161.
German silver, poisoning by, 475.
Gilmour, case of, 372.
Glanders poison, no effect by the stomach, 36.
Glass, powdered, injurious effects of, 33.
Glaze, lead-, poisoning by, 446.
Gloeckler, case of, 363.
Godfrey's cordial, 539.
Gold, poisoning by, 495.
Good, case of, 156.
Goulard's extract, 430; water, 430.
Grain, arsenic in, 373; copper in, 474.
Gray and *Bright*, case of, 116.
Green vitriol, 496.
Greene, case of, 696.
Groats poisoned by lead, 450.

Habit, its influence on organic poisons, 91; no influence on mineral poisons, 97.
Hæmatemesis, diagnosis of, 129.
Hair, a mechanical irritant, 32; alleged presence of arsenic in the, 51.
Hair-dyes, composition of, 437, 451.
Hanging or poisoning, death from, 214.
Harris, *Mr.*, case of, 570.
Hartley, case of, 27, 188.
Hart, *Sarah*, case of, 601.
Hartshorn, spirits of, poisoning by, 302.
Haydon, case of, 22, 303.
Haywood, *Mr.*, case of, 254.
Heart, diseases of, mistaken for poisoning, 145.
Hedger, case of, 217.
Helleboraster, 508.
Hellebore, poisoning by, 21, 508.
Helleborus fœtidus, 509.
Helleborus niger, 508.
Helvella esculenta, 666.
Hemlock, poisoning by, 708.
Hemlock, water-dropwort, 713.
Henbane, poisoning by, 667.
Heritier, case of, 581.
Hernia, strangulated, 126.
Hierapicra, 21.
Higgins, case of, 197.
Hocussing, by cocculus indicus, 663; by datura, 737.
Hoffmann's liquor, 648.
Honey, poisoned, 202.
Hong-kong bread, 342.
Horseradish, root known from aconite, 724.
Horses, action of arsenic on, 93.
Hume, case of, 439.
Humphreys, case of, 113.
Hunter, case of, 49, 154, 177, 364.
Hydrogen, arsenuretted, poisoning by, 395.
Hydrogen-test for arsenic, 353.
Hydrochloric acid, 265.
Hydrochlorate of morphia, 545; of strychnia, 691.
Hydrocyanic acid, 562. See PRUSSIC ACID,
Hyoscyamia, 669.
Hyoscyamus niger, poisoning by, 667.
Hysteria, tetanic convulsions in, 136.

Iatropha manihot, 626.

Idiopathic asphyxia, 145.
Idiosyncrasy in poisoning, 101.
Ignatia amara, 677.
Ileus mistaken for poisoning, 126.
Imbibition cadaveric, 68.
Imputed poisoning, 122.
Indian tobacco, poisoning by, 731.
Indigo, sulphate of, 250.
Infants, poisoning of, by opium, 91, 133, 533.
Inspection of the body, rules for the, 167.
Insurance, life-, opium-eating in cases of, 93.
Internal strangulation mistaken for poisoning, 127.
Intestines, obstruction of the, 126; perforation of the, 126.
Intolerance of medicines, 109.
Intoxication, influence of, on poisons, 109; from alcohol, 635; distinguished from concussion, 638.
Intussusception, mistaken for poisoning, 126, 128, 210.
Ipecacuanha, compound powder of, 541.
Iron, metallic, not poisonous, 29; poisoning by the preparations of, 495.
Irritant poisons, 104, 153; mineral, 319; animal and vegetable, 499.
Irritants, mechanical, 27.
Irritant poisoning, diseases resembling, 123.

James's powder, 488.
Jargonelle pear, artificial essence of, 619.
Java poison, 678.
Jennings, case of, 157.
Johnson, case of, 377.
Joyce, case of, 143.
Juniperus sabina, 500.

Kernels, cherry and peach, poisoning by, 624.
Ketchup, alleged poisoning by, 667.
King's yellow, alleged death from, 209.
Kremser white, 431.

Laburnum bark and seeds, poisonous effects of, 739.
Lace bleached by lead, effects of, 433.
Lacoste, case of, 50, 331.
Lactuca virosa and sativa, 670.
Lactucarium, 670.
Lactucin, 670.
Laffarge, case of, 193, 367, 373.
L'Angelier, case of, 97, 187.
Lapis infernalis, 495.
Laudanum, large doses of, 98; poisoning by, 519.
Laurel oil and water, 621.
LEAD, absorption of, 65; poisoning by the salts of, 374; symptoms, 429; subacetate of, 430; carbonate of, 431; sulphate of, 432; chromate of, 432; chronic effects of, 432; oxides of, 432; water poisoned by, 436, 451; external application of, 437; appearances after death, 438; fatal dose, period of death, 439; treatment of poisoning by, 440; chemical analysis, 441; in organic mixtures, 443; in the tissues, 444; quantitative analysis, 444; in articles of food, 446; in milk, beer, cider, 447; in pork, in flour, 448; in sugar and snuff, 449; in oatmeal, 450; in water, 451; in herbage, 455; cattle poisoned by, 455.
Lead-foil for wrapping food, 450.
Lead-glaze poisonous, 446.
Lead-shot, poisoning by, 427; wine poisoned by, 432, 448.
Lettuce-opium, 670.
Lettuce, strong-scented, effects of, 670.
Levant-nut, 662. See COCCULUS INDICUS.
Lever, case of, 347, 363.
Ligustrum vulgare, 742.
Life-insurance, opium-eating in cases of, 93.
Liqueurs, poisoning by, 624.
Liquids, boiling, effects of, when swallowed, 33.
Litharge, poisoning by, 432.
Lobelia inflata, poisoning by, 731.
Local action of poisons, 80.
Lofthouse, case of, 322.
Lolium temulentum, 664.
Lozenges, opium, poisoning by, 542.
Lucas and *Reader*, case of, 380.
Lucifer matches, poisoning by, 312.
Lumbrici, 166.
Lunar caustic, 495.
Lungs, action of poisons by the, 37.
Lytta vesicatoria, 511.

McConkey, case of, 720.
Macormick, case of, 344.
McCracken, case of, 338.
McMullen, case of, 61.
Macquer's salt of arsenic, 393.
Madagascar poison, 678.
Magistery of bismuth, 497.
Magnesia, sulphate of, effects of, 19.
Maher and *Lynam*, case of, 321.
Malaret, case of, 380.
Male, Dr., case of, 721.
Malignant cholera mistaken for poisoning, 119.
Manihot jatropha, 626.
Mar, Earl of, case of, 524.
Marcooley, case of, 565, 576, 596.
Marsh's process for arsenic, 353; delicacy of, and corroborative experiments relating to, 357.
Matches, lucifer, poisoning by, 312.
Meadow saffron, poisoning by, 505.
Mechanical irritants, 27.
Meconic acid, 551.

Medicines, saline, poisonous, 19; poison substituted for, 115.
Menispermum cocculus, 662.
Mercurius vitæ, 485.
Mercury, absorption of, 67.
MERCURY, liquid, not poisonous, 28, 397; poisoning by the vapor of, 406; bichloride of, 398; salivation from, 402; chloride of, 421; ammonio-chloride of, 422; red oxide of, 423; subsulphate of, 425; nitrates of, 426; cyanide of, 424, 634.
Merritt, Anne, case of, 53.
Mercurial medicines, death from, 403.
Metallic irritants, 319.
Metalloidal irritants, 311.
Metallic poisons, 319.
Metals, pure, not poisonous, 29.
Methylated spirit, 641.
Milk, poisoned, 201.
Mineral green, 329; turbith, 425.
Mineral solution, Fowler's, 383.
Minium, 432.
MONKSHOOD, poisoning by leaves and extract of, 718; root mistaken for horseradish, 719; poisoning by tincture of, 721; analysis, 723.
Montgomery, case of, 596, 603.
Morchella esculenta, 666.
MORPHIA, absorption of, 73, and its salts, poisoning by, 543; symptoms, 543; appearances, 544; fatal dose, 545; acetate, 546; tests for, 549; in organic liquids, 556; in the tissues, 560.
Mouth, cancer of the, 405.
Munn, case of, 212.
MURIATIC ACID, poisoning by, 265; symptoms, 265; appearances after death, 267; fatal dose, 268; treatment, 269; analysis, 270; in organic liquids, 271; on clothing, 273.
Muriate of iron, 496; of soda, 19; of morphia, 546; of strychnia, 691.
Murton and *Petch*, case of, 93.
Mushrooms, poisonous, 665.

Narceine, 553.
Narcotic poisoning, diseases resembling, 129; treatment of, 535.
Narcotic poisons, 518.
Narcotico-irritant poisons, 705.
Narcotina, 548; chemical properties of, 552.
Needles, effects of, when swallowed, 30.
Neurotic poisons, 105, 155, 518.
Newton, Reg. v., 196, 378.
Nickel silver, poisoning by, 476.
Nicotiana tabacum, 657.
Nicotina, effects of, 660; analysis, 661.
Nightshade, woody and garden, 670; deadly, 725.
Nitrate of potash, 306; of mercury, 426; of silver, 495; of bismuth, 497.
Nitre, 306.
NITRIC ACID, poisoning by, 252; vapor of, 252; symptoms, 252; in diluted state, 253; appearances after death, 255; chronic poisoning by, 257; fatal dose, 257; period of death, 258; treatment, 259; chemical analysis, 259; tests, 260; in organic liquids, 261; absorption and elimination of, 262; on clothing, 264; quantitative analysis, 264.
Nitrobenzine, effects of, 618.
Nitrobenzole, 618.
Non-metallic irritants, 223.
Normal arsenic, 371; copper, 471.
North, case of, 189.
Noxious thing, signification of, 29.
Noyau, 624.
NUX VOMICA, poisoning by, 673; symptoms, 673; chronic poisoning by, 674; appearances and fatal dose of, 675; analysis, 676.
Nux vomica, extract of, 674.
Nux vomica bark, 677.

Oatmeal poisoned by lead, 450.
Obstructed intestine, death from, 126.
Œnanthe crocata, 713.
Œsophagus, perforation of, 162.
Oil, boiling, effects of, 34; croton, 503; of savin, 503; of laurel, 621.
OIL OF BITTER ALMONDS, conversion of, in the blood, 87; prussic acid contained in, 606; effects of the vapor, 606; symptoms of poisoning by, 607; appearances after death, 609; fatal dose, 613; period of death, 615; local action, 615; chemical analysis, 616; in organic liquids, 617; deprived of prussic acid, effects of, 618.
Oil of vitriol, 223.
Oorara poison, 679.
Opinions, premature, danger of, 191.
OPIUM, influence of habit on, 91, 532; large doses of, in tetanus, 99; poisoning by, 519; symptoms caused by, 519; period of commencement, 522; remission of, 523; chronic poisoning by, 524; effects of external application, 525; appearances after death in, 527; fatal dose, accumulative properties of, 529; recovery from large doses, 530; action of, on infants, 532; period of death, 534; treatment, antidotes, 535; alkaloids in, 543, 548; analysis, 549; in organic mixtures, 553; opium not detected, 553; quantitative analysis of, 561; proportion of, in medicinal preparations, 561.
Opium-eating, 524; in reference to life-insurance, 93.

Opium-lozenges, 542.
Opium, wine of, 542.
Orpiment, 393; white arsenic converted to, in the dead body, 182.
Orange-peel, suspected death from, 33.
Ordeal poison bean of Africa, 678.
Oxalate of lime, 291.
Oxalic acid, poisoning by, 276; symptoms, 277; appearances after death, 279; fatal dose of, 282; recoveries from large doses, 283; period of death, 284; treatment, 285; chemical analysis, 285; in organic liquids, 288; in vegetables, 291; absorption of, 293; quantitative analysis; 294.

Paint, poisoning by, 434.
Painters' colic, 433.
Palmer, Ann, case of, 99.
Palmer, Walter, case of, 565, 597, 603.
Palmer, William, case of, 114, 137, 185, 697.
Papaver somniferum, 519.
Paper hangings, arsenical, 388, 743.
Papier Moure, 393.
Paralysis from lead, 435, 437; from mercury, 406.
Paramorphine, 549.
Paregoric elixir, 540.
Parisian epileptics, cases of the, 566.
Parsley, fool's, poisoning by, 716.
Peach-kernels, poisoning by, 626.
Peach-nut oil, 605.
Pearl-ash, poisoning by, 296.
Pearl-white, 497.
Percussion caps, poisoning by, 32.
Perforation of the stomach mistaken for poisoning, 126; causes of, 158; spontaneous, 161; of œsophagus and intestines, 163; by foreign bodies, 164; by worms, 164.
Peritonitis, 125.
Persulphuret of mercury, 424.
Phellandrium aquaticum, 716.
Phosphoric and phosphorous acids, action of, 274.
Phosphorus paste, nature of, 318.
Phosphorus, poisoning by, 311; vapor of, chronic poisoning by, 313; appearances after death, 313; absorption of, 314; fatal doses of, 315; period of death, 316; chemical analysis, 316; non-detection of, 179.
Phosphorus, red or amorphous, not poisonous, 318.
Pickles poisoned by copper, 474.
Picrotoxia, 664.
Pie-plant, alleged poisoning by, 292.
Pine-apple, artificial essence of, 619.
Pins and needles, injurious effects of, when swallowed, 30.
Pigment, green, poisoning by, 329.
Placenta, poisons detected in the, 55.
Poison, definition of, 18, 23; legal meaning of, 25; evidence from the quantity of, in the dead body, 183; proof of administration of, 188; annual deaths from 221.
Poisons, absorption of, 35 (see Absorption); detected in the blood, 38; elimination of, 44; detection of, in the fœtus and in entozoa, 55; local effects, 80; mode in which they destroy life, 85; changes of, in the blood, 87; influence of habit on, 91; tolerance of, 98; effect of idiosyncrasy, 101; classification of, 102; substituted for medicine, 115; period of death from, 149; accumulative, 152; causes of non-detection of, 174; decomposition of, in the living body, 178; loss of, in the dead body, 181; in tests and apparatus, 190, 195; mineral irritant, 223; vegetable and animal irritant, 499; narcotic, narcotico-irritant, 518.
Poisoning, statute relative to, 26; evidence of, in the living subject, 106; disease mistaken for, 112; ambiguous cases of, 120; feigned and imputed, 122; rules for investigating cases of, 148; evidence of, in the dead body, 149; acute and chronic, 151; evidence of, from chemical analysis, 169; from experiments on animals, 195; suspected cases of, 204; compound; 215; statistics of, 219.
Poisoned animals, flesh of, 200.
Poisoned wounds, 38.
Poppies, poisoning by, 537.
Pork, lead in, 448.
Porphyroxine, 553, 556.
Port, case of, 333.
Port-wine poisoned by arsenic, 340; by lead-shot, 448.
Porter, cocculus indicus in, 663.
Potash and its carbonates, poisoning by, 296; analysis of, 298; absorption of, 299; binoxalate of, 304; bitartrate of, 305; nitrate, 306; sulphate, 308; arsenite, 383; arseniate, 392; bichromate of, 498.
Potassium, cyanide of, 630; sulphocyanide of, 632; ferrocyanide of, 633.
Poudre d'Italie, 437.
Pounded glass, injurious effects of, 33.
Pralet, case of, 580.
Praslin, Duke of, case of, 47, 125, 324, 344.
Premature opinions, 191.
Privet berries, poisoning by, 742.
Prussian blue, 634.
Prussic acid, absorption and elimination of, 71; poisoning by, 562; effects of the vapor, 563; symptoms caused by, 564;

taste and odor of, 567; period of commencement of symptoms, 568; power of volition and locomotion in cases of poisoning by, 568; its effects contrasted with those of opium, 572; chronic poisoning by, 572; external application of, 573; appearances after death, 574; loss of odor in the dead body, 575; fatal doses, 583; recovery from large doses, 584; period of death, 586; treatment, 587; chemical analysis, 589; tests for the liquid and vapor, 591; detection of, in organic liquids, 593; detection in the tissues, 594; period at which, detected after death, 595; alleged spontaneous production of, 598; not detected in the body, 603; changes of, in the dead body, 603; quantitative analysis, 604; leaves and kernels containing, 624.
Ptyalism (salivation), from mercury, 403.
Putrefaction, loss of poisons by, 181.
Pyroxylic spirit, 641.

Quantity of poison found in the body, evidence from, 183, 186.
Quicksilver, see Mercury, 397.

Ramus, case of, 580.
Ratafia, poisoning by, 624.
Realgar, 393; used in quack preparations, 393.
Red arsenic, 393.
Red lead, 432.
Red oxide of mercury, 423.
Red phosphorus, 319.
Red precipitate, 423.
Red spirit of nitre, 252.
Redness of mucous membrane from poisoning and disease, 154; destroyed by putrefaction, 156.
Reinsch's process for arsenic, 358; delicacy of, 360, 743.
Remote action of poisons, 83.
Rhubarb, garden, alleged poisoning by, 292.
Rhymes, case of, 337.
Ribstone pippin, artificial essence of, 619.
Rice, death from, 33.
Richards v. *Cocking*, case of, 494.
Richardson, case of, 372.
Rodanbosh, case of, 21.
Roada, case of, 227.
Rules for investigating cases of poisoning in the living, 148; in the dead, 167.
Rum, poisoning by, 636.
Rupture of stomach, 147; of the gall-bladder, 147.
Russell, case of, 150.
Rye bread, noxious effects of, 665.

Sabina juniperus, 500.
Sadleir, Mr., case of, 187, 612.
Saffron, meadow, 505.
Sagar, case of, 329.
Sager, case of, 322.
Saint Ignatius's bean, 677.
Sal de duobus, 308.
Sal prunella, 306.
Sal polychrest, 308.
Sal volatile, 302.
Saline medicines poisonous, 18.
Salivation, mercurial, 403.
Salts, alkaline, poisoning by, 303.
Salt, common, death from, 19.
Salt of sorrel, 304.
Saltpetre, 306.
Salt, spirits of, 265.
Salts, Epsom, fatal effects of, 20.
SAVIN, poisoning by, 500; appearances, 502; analysis, 502; oil of, 503.
Scheele's green, 385; prussic acid, 563.
Schwabe, case of, 229.
Schweinfürth green, 385.
Secale cornutum, 665.
Sedative solution, Battley's, 542.
Seed corn with arsenic, 373; with copper, 474.
Serpent poison (echidnine), 36.
Sesquicarbonate of ammonia, 302.
Sesquichloride of antimony, 489.
Sesquisulphuret of arsenic, 388.
Shaking palsy from mercury, 402.
Sheepwash, poisoning by, 384.
Sherrington, case of, 198.
Shot-lead, poisoning by, 432, 448.
Silver, nitrate of, 41; absorption of, 65; poisoning by, 495; cyanide of, 634.
Skin, action of poisons through the, 35, 334, 344.
Slow poisoning, 328, 330.
Sleep, influence of, on poisons, 108.
Smalt, arsenic in, 373.
Smith, Azenath, case of, 695.
Smith, Madeline, case of, 97, 187, 328.
Smith, Reg. v., 426, 466.
Smithers, cases of the, 565.
Smyth, Mrs., case of, 683.
Smoking, noxious effects of, 659.
Snakewood, 677.
Snuff poisoned by lead, 449.
Soap-lees, poisoning by, 297.
SODA, muriate of, 19; and its carbonates, poisoning by, 297.
Sodium, chloride of, 19.
Softening of mucous membrane from poison and disease, 158.
Solomon v. *Lawson*, case of, 120.
Solania, 671.
Solanum dulcamara and nigrum, 670.
Sorrel, salt of, 304.
South American poison, 679.
Southgate, case of, 27.
Spanish flies, poisoning by, 511.
Sphacelia segetum, 665.

Spinal marrow, diseases of, 144.
Spinal poisons, 103, 164, 672.
Spirit dyers', 495.
Spirits, ardent, effects of, 635.
Spirit of salt, 265.
Spirits of hartshorn, 299.
Sponge, injurious effects of, 32.
Spontaneous perforation, 161.
Spotted hemlock, 708.
Spurred rye, 665.
Stafford, Mr. A., case of, 523, 536.
Starch, alleged presence of arsenic in, 373.
Statistics of poisoning, 219.
Stewart, case of, 560.
Stibiated tartar, 476.
Stomach, absorption of poisons by the, 36; death from distension of, 146; rupture of, 147; perforations of, 158.
Stramonium, poisoning by, 735.
Strangulated hernia, 126.
Strangulation, internal, death from, 126.
Strangulation or poisoning, death from, 214.
STRYCHNIA, absorption of, 74; local action of, 82; poisoning by, mistaken for tetanus, 140; and its salts, poisoning by, 681; symptoms, 681; period of commencement, 682; chronic poisoning by, accumulative properties of, 684; appearances after death, 685; fatal dose, 687; period at which death takes place, 687; treatment, 688; chemical analysis, 690; tests for, 690; in organic mixtures, 692; the poison not detected, 694; in the tissues, 694; trials for murder by, 695.
Strychnos nux vomica, 672; tieuté, 678; toxifera, 679; colubrina, 677; ignatii, 677.
Subacetate of copper, 461; of lead, 430.
Subchloride of copper, 462.
Sublimate, corrosive, 398.
Subnitrate of bismuth, 497.
Sulphate of mercury, 425.
Sudden death, causes of, 150.
Suffocation or poisoning, death from, 216.
SUGAR OF LEAD, symptoms caused by, 428; chronic poisoning by, 432; appearances, 438; fatal dose and period of death, 439; treatment of poisoning by, 439; analysis of, 441; in organic mixtures, 443; detection of, in the tissues, 444.
Sugar poisoned by lead, 449.
Sulphate of magnesia, 19; of indigo, 250; of potash, 308; of copper, 459; of zinc, 491; of iron, 495; of morphia, 543, 545; of strychnia, 687, 691.
Sulphindigotic acid, 250.
Sulphocyanic acid, 632.
Sulphuret of mercury, 424.
Sulphuret of arsenic, death from, or disease, 209; local action of, 335; poisoning by, 388.
SULPHURIC ACID, symptoms caused by, 224; period of commencement, 226; acts of volition and locomotion in poisoning by, 229; remission and intermission of symptoms, 230; appearances after death from, 231; chronic poisoning by, 235; absorption and elimination of, 237; fatal dose of, 238; period of death, 239; treatment of, 240; chemical analysis of, 242; tests for, 242; analysis in organic liquids, 244; stains of, on clothing, 247; aromatic, poisoning by, 249; quantitative analysis, 250.
Symptoms, evidence from, in poisoning, 113, 123; rules for observing, 148.
Syncopal asphyxia, 145.
Syrup of poppies, 538.
Systemic action of poisons, 83.

Tanghin poison, 678.
Tanghinia venenifera, 678.
Tape-worm, 167.
Tapioca-plant poisonous, 626.
Tartar, cream of, action, 305.
TARTAR EMETIC, tolerance of large doses of, 99; symptoms and appearances, 477; chemical analysis, 483; arsenic contained in, 488.
Tartaric acid, 294.
Tartarized-antimony, 476.
Tasteless ague drop, 383.
Tawell, case of, 579, 584, 601.
Taxus baccata, 740.
Tetanic convulsions, natural causes of, 138.
Tetanus, large doses of opium in, 98; symptoms of, 136; hysterical, 136; mistaken for poisoning, 138.
Tests and apparatus, 190; fallacies respecting the use of, 356.
Thebaina, 549.
Thomas, case of, 231, 234, 364.
Thornapple, poisoning by, 735.
Ticunas poison, 679.
Tieuté, upas, 678.
Tin, poisonous preparations of, 495.
Tinfoil, patent, noxious effects of, 450.
Tissues, poisons in the, 43, 55.
Tobacco, poisoning by, 657.
Tobacco, Indian, effects of, 731.
Toffana aqua, 177.
Tolerance of poisons, 98.
Trades subject to painters' colic, 433.
Treacle, lead in, 449.
Trisnitrate of bismuth, 497.
Truffles, poisoning by, 666.
Turbith mineral, 425.
Turners, case of the, 328.

Ulceration of mucous membrane from poisoning and disease, 156.
Upas, antiar, 678.
Upas poison of Java, 678.
Upas tieuté, 678.
Urari poison, 679.
Urine, poisons detected in the, 44.
Uterus, arsenic detected in the, 55.

Vapors of arsenic, alleged death from, 120, 216, 382; of nitric acid, death from, 254; of ammonia, effects of, 299; of phosphorus, 313; of mercury, effects of, 406; of prussic acid, 563.
Vegetable irritants, 499.
Vegetable acids, 276.
Venom of serpents, 36.
Veratria, poisonous effects of, 510.
Veratrum album, 510.
Verdigris, 461.
Vermilion, 424.
Vinegar, arsenic in, 372.
Vinum antimonii, 476; opii, 542.
Violence, death from, mistaken for poisoning, 213.
Viscera, removal of, in the dead body, 167; cautions respecting the preservation of, 193.
Vitriol, oil of, 223; blue, poisoning by, 21, 459; white, 491; green, 496.
Vitriolic acid, 223.
Volatile poisons, absorption and elimination of, 71, 79; changes in, 182.
Vomited matters, detection of poison in, 122.

Wainwright v. *Bland*, 135.
Wall papers, poisonous, 387, 743.
Walsh, case of, 398, 421.
Waring, case of, 344.
Warner, Dr., case of, 687.
Water, effects of cold, 25; of boiling, 34; of wells accidentally poisoned, 121; poisoned lead, 436, 451; by copper, 473.
Water, bitter almond, poisoning by, 618.
Water-hemlock, 711, 716.
Water, Laurel, 621.
Watkins, Reg. v., 294.
Welch, case of, 408.
Whisker, case of, 20.
Whiskey, poisoning by, 635.
White, case of, 21.
White, hellebore, 20, 508.
White lead, 431.
White precipitate, 26; poisoning by, 422.
White vitriol or copperas, 491.
Wine poisoned by arsenic, 340; by lead-shot, 448.
Wine, antimonial, 476.
Wine of opium, 542.
Wishart, case of, 367.
Wolfsbane, poisoning by, 718.
Wood-sorrel, salt of, 304.
Woody nightshade, 670.
Wooler, case of, 96, 108, 329.
Woorara poison, 679.
Worms, poisons found in, 55; perforations of the intestines by, 164; species of, 166; alleged death from, or poison, 208.
Wounds, poisoned, 38.
Wourali poison, 679.
Wren, Reg. v., 673.

Yellow arsenic, 393.
Yellow, King's, alleged poisoning by, 209.
Yellow sulphate of mercury, 425.
Yew-berries and leaves, poisoning by, 740.

ZINC, sulphate of, 491; chloride of, 492; symptoms and appearances, 492; analysis, 494; carbonate of, 495.

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